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# THE BOSTON Medical and Surgical JOURNAL

VOLUME 196

APRIL 21, 1927

NUMBER 16

## ORIGINAL ARTICLES

### THE DIAGNOSIS OF ABDOMINAL EMERGENCIES\*

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THE diagnosis of acute abdominal disorders which demand prompt surgical intervention presents an interesting and fascinating problem to the medical mind. No other field of medicine throws the doctor so completely back upon his own resources, those resources which dwell within himself and are manifested by the exercise of his special senses and reason. In these days of elaborate laboratory diagnoses, when all the facilities of modern science in its many ramifications are called into play, it is rather exhilarating to throw away all outside aids, all so-called exact methods and instruments of precision, to grapple with the problem unaided and strive to deduce its correct solution by the proper arrangement of facts obtained by one's own observations, and correlated by one's own experiences. We are no longer interpreting chemical reactions, reading instrumental findings, or puzzling over photographic films, but we are noting nature's reaction to abnormal stimuli within the human body,—reactions which, although they may be constant, are so influenced by individual conditions and multiplicity of contributing factors that each diagnosis is an independent entity.

In correctly determining the cause of any acute abdominal disturbance, one can discard, and usually must discard, any aid other than that furnished by his own special senses. Even the clinical thermometer is more or less useless. The employment of one's special senses, even, is restricted, for sight, touch, and hearing only are used. A second factor enters into the interest of the problem and that is the element of time. Diagnosis is merely a means to an end and this end is the successful treatment of the determined lesion. In acute abdominal conditions, the favorable outcome is usually brought about by prompt diagnosis and speedy surgical intervention. This necessity for rapid action is best exemplified in a case of ruptured peptic ulcer, where literally moments count.

In making a diagnosis of an acute abdominal lesion, one is usually deprived of the aid of counsel, and the responsibility is one's own and

cannot be shared with others. The diagnosis made, one is then often confronted with the necessity of convincing the patient, his friends or relatives, of the need of prompt action,—not always an easy matter. Early in my career I made a diagnosis of acute appendicitis in a young man and urged immediate operation, only to meet the unalterable determination of his father expressed by the words, "No one in our family has ever been operated yet and we aren't going to begin now."

I realize in choosing my subject tonight, that I can contribute nothing new to your knowledge of acute abdominal conditions, but I feel that often much can be gained by pausing a while to review and thoughtfully analyze our conception of certain conditions. I further realize that exceptions can be taken to almost any statement that I may make. One can nearly always recall a case. It is not one case, or even several, which constructs the background for the formation of tenable opinions. The life insurance actuary cannot foretell the life expectancy in a given case, but he can very accurately foretell it in a sizeable group of such cases. In considering signs and symptoms one is dealing with averages, and wide deviations may be encountered.

It is possible to read an accurate description of several or many cases of a given abdominal condition and yet be unable to recognize it when first encountered. Nothing can take the place of clinical experience, not even the best of teaching. Reading and instruction are valuable in that they present to us accumulated knowledge and decrease the necessity for original observations and research which most of us are incapable of making or conducting.

In following some of our great clinicians at their work of diagnosing acute abdominal conditions, the uninitiated often consider that they are superficial in their histories and examinations, only to find that the secret of their greatness lies in the ability to pick the important facts and disregard those which contribute nothing to the problem. In these days of exact methods, it seems foolish to emphasize the necessity of observing the patient, yet much can be learned by merely standing and looking at him.

\*Read before the Fall River Medical Society, Fall River, January 12, 1927.

The knowledge to be gained in this fashion is hard to describe, yet it is very real. The flushed face and exaggerated movement of the *alae nasi* are sometimes the determining factors which lead us to postpone the appendectomy in a child to prove later that he has pneumonia. You all know that the creation of Sherlock Holmes was prompted by Conan Doyle's admiration of a Scotch physician's almost uncanny powers of observation and resulting deductions. We hear much of the prowess of the old time family doctor and what he could learn of a patient by observation. Draper of New York, has attempted to scientifically record and analyze the facts which may be obtained from observing a patient. Whether or not we have any faith in his conclusions or believe in his ulcer types, gall bladder types, and so on, we certainly must admit that he seems to have found a little something which may lead to greater things.

Pain is a constant factor in acute abdominal conditions and merely as pain it means nothing, as it is common to all. Its mode of onset, duration, location, and possible control are of the utmost importance, while its character as described by the patient may or may not be of value. We often hear pain described as boring, lancinating, cutting, sharp, grinding, and so on. Describing a sensation is a difficult matter and something which few people can do with any great degree of accuracy. Again, susceptibility to pain is a variable factor and that which seems unbearable to one may be borne by another with equanimity. I have known of a man with a ruptured peptic ulcer driving his own automobile eighteen miles to his home and then calling his family physician on the telephone. Another man, apparently similarly stricken, is prostrated at once. It is hard for most patients to definitely locate a pain and the best that they can do is to assign it to a position high up, low down, on the right, or on the left of the abdomen. Ask a patient with appendicitis where he has a pain and he will rather vaguely move his hand over the right lower quadrant and if you insist on greater accuracy, he will poke himself until he finds the point of greatest tenderness, thus really confusing pain with tenderness on pressure. The same reaction will be found relative to the gall bladder. Unless especially urged, the patient will pass his hand over the entire epigastrium with possibly slightly more emphasis on the right side. The look and actions of a patient may give us some insight into the severity of his pain.

Vomiting is of little value as a diagnostic aid in acute abdominal conditions, as it may be present with any acute lesion or it may be absent. In most cases it seems to be a reaction to pain, and as patients vary in their other reactions to this stimulus, so they vary in their tendency to vomit.

A rise in temperature, or fever, is an accom-

paniment of inflammation and denotes an absorption of toxic products of this inflammation. Many of the acute abdominal lesions are not inflammatory in nature at their onset and any inflammation associated with them is secondary. Furthermore, although some of the lesions may be inflammatory, there may be little absorption and, therefore, little or no temperature. In the subject under consideration, a rise in temperature is an indication of the extent to which the process has progressed, rather than an aid in the diagnosing of it. With rupture of a viscus, there is no temperature until the secondary peritonitis develops, a condition which one is seeking to forestall. A gangrenous appendix may develop without an accompanying temperature. A temperature, if present, may mean something, but its absence is no premise on which to base a line of reasoning.

Leucocytosis is usually considered when making a differential diagnosis of acute abdominal lesions, but in our experience it is of doubtful aid. Just as a rise in temperature is an accompaniment of inflammation, so is an increase in the white cells circulating in the blood stream an indication of inflammation. It is present with peritonitis but absent with the initial lesion causing it. Gangrene may or may not call forth the white cells. The late Albert Oschner in discussing a paper on appendicitis said, "At the Augustinia Hospital we always have a white count in a suspected case of appendicitis, but we never look at it until after the operation." The majority of acute abdominal cases are seen at the patient's home and our diagnosis must be made without recourse to blood findings, even though they might be of value. In considering leucocytosis many men lay great stress upon the differential count and argue much from the index of resistance. In the past twenty-five years the hematologist has exactly reversed his conception of the index of resistance, and its most ardent advocates claim only for it, prognostic value. This value, of course, is for untreated cases, and when dealing with acute abdominal conditions most of us are not going to leave them untreated for any length of time.

One often hears of the abdominal pulse, but the rate and character of the pulse are indications of the patient's general condition rather than factors in determining his pathological lesion.

The age of a patient is often an aid in determining his lesion, but like other factors, it cannot be given too great weight, and oftentimes our preconceived opinions may be shattered. Appendicitis is called the disease of young adult life, and yet it may occur at any age. We have had several cases in patients over seventy years of age. Cholecystitis, we are told, is a disease of middle life, but we had one case of gangrenous gall bladder with stones in a girl of fourteen years of age. Our pre-operative diagnosis was



appendicitis. We usually consider carcinoma as a disease occurring in people past middle life, yet in the past three months we have seen two cases which confirm our previous opinion that you can never rule out the possibility of carcinoma on age alone. The first case was one of obstruction of the bowels in a boy of fourteen years of age. Operation disclosed an adenocarcinoma of the transverse colon. The second case was also one of obstruction of the bowels in a girl of eighteen years of age. The obstruction was in the sigmoid flexure of the colon, caused by proximity to and involvement with an adenocarcinoma of the left ovary. In both of these cases the pain was in the epigastrium, thus refuting Dr. Jones' observation that the pain of large bowel involvement is always below the umbilicus.

In making a diagnosis of an acute abdominal condition, one must rely entirely upon the history of the case, one's observation of the patient, and what one may glean from physical examination, particularly from palpation.

In considering differential diagnosis of an acute abdominal lesion, one has a rather formidable list of conditions to bear in mind, although some of them may be dismissed with rather scant attention, but one must always remember that there may be a joker in the pack. Some very wonderful and unexpected conditions may develop in the abdomen and it is because of these unexpected conditions and the uncertainty of the diagnosis in general, that we emphasize the necessity of experience and training in surgery. The surgeon who operates on an acute abdomen, must be exceptionally well informed, and be able to perform a multiplicity of operations. He never can be absolutely sure of what he is to find or what he may be called upon to do. Our differential list is somewhat as follows:—

Appendicitis, obstruction of the bowels, acute cholecystitis, rupture of a peptic ulcer, acute pancreatitis, diverticulitis, mesenteric thrombosis, inflammation of Meckel's diverticulum, ectopic gestation, ovarian cyst with twisted pedicle, infected or strangulated fibroids, typhoid perforation, injury to a viscus, acute salpingitis, ureteral calculus, pyelitis, and several medical conditions.

It is not my intention to take up the consideration of medical conditions which may occur in the abdomen, or which may simulate abdominal disease, as you have recently listened to a paper on this important phase of the subject. I do wish, however, to emphasize the necessity of being conversant with these diseases and their manifestations. Every abdominal surgeon should have a very thorough training in internal medicine, and should keep abreast of its advances.

The last three lesions mentioned in our differential list, namely, acute salpingitis, ureteral calculus, and pyelitis are given, not because they

are acute surgical conditions, but because they all may closely simulate appendicitis, and often they must be ruled out in our diagnosis. They are not surgical in the sense that they require operation, with the possible exception of ureteral calculus, and here there is no urgency, and oftentimes a cure may be successfully effected without recourse to the scalpel. You will further note that I have included in our list a number of conditions claimed by the gynecologists as their own. These pelvic lesions may have abdominal symptoms and as acute conditions they usually come under the observation of the general surgeon, except possibly in a large well organized hospital. Many times it is difficult to draw the line between surgery and gynecology, unless one accept Dr. Blake's definition that, "Gynecology is any operation on a woman."

Acute salpingitis is nearly always bilateral, and the resulting pain and tenderness is, therefore, on both sides of the lower abdomen. Rigidity or spasm of the abdominal muscles is usually slight, and vaginal examination aids materially in the diagnosis.

Pyelitis, like several other lesions of the urinary tract, is more often right sided than left. It is a fairly common condition in women and children, and is often confused with appendicitis. It differs from the latter, in that the pain is more diffuse over the right side. There is little or no muscular spasm or rigidity, and there is no definite localizing tenderness.

Ureteral calculus on the right side, may very closely resemble pyelitis in its symptoms, and therefore, differs from appendicitis in the same respects. The pain with calculus may be more intense than that with appendicitis, and the patient is bathed in perspiration, especially over the upper lip. This last condition is rather characteristic of ureteral calculus.

Appendicitis is by long odds the most common affection of the abdomen and it may simulate several of the other abdominal lesions, because of the variation in the symptoms by which it manifests itself. It is common knowledge that the pain of appendicitis often originates in the epigastrium, or about the umbilicus, but sooner or later it localizes itself in the right lower quadrant of the abdomen. Spasm or rigidity of the right rectus muscle is often present, but it may be wanting. We have been especially struck by the fact that it is very often absent with severe involvement of the appendix, especially with gangrenous appendicitis and when the appendix is within the pelvis. Deep pressure over the left lower quadrant of the abdomen will often cause pain on the right side with appendicitis. Occasionally firm, deep, continued pressure over the appendix, suddenly released, will cause acute pain. The deep pelvic appendix, when diseased, may produce symptoms of bladder irritation. We believe that the cardinal sign of acute appendicitis, the sign to be valued above all others,

is tenderness on pressure over McBurney's point. Tenderness is present at this point, no matter where the appendix may lie within the abdomen. We further believe that it is impossible to judge the severity of the pathological condition present. All we can say is that it is a case of appendicitis, and this being true, the sooner the offending organ is removed, the better.

All that I have said concerning the signs and symptoms of appendicitis may be reversed in the case of a left sided appendix. In the few cases of which we have personal knowledge, there has been a transposition of all of the viscera, so that auscultation of the heart has been a valuable sign pointing to a correct diagnosis. Carman says that it is possible to have transposition of the colon without transposition of the other viscera, but we have no personal knowledge of such a condition.

One of the abdominal catastrophes which closely resembles appendicitis in its signs and symptoms, and which is often mistaken for it, is rupture of a peptic ulcer. Many men contend that rupture of a viscus without contributing injury occurs only in an organ weakened by a chronic lesion. This is probably true, but under the stress of the acute trouble, it is often impossible to elicit a history of long standing. This is particularly true in cases of rupture of a peptic ulcer. It is almost inconceivable that an ulcer can progress to perforation without antecedent symptoms, and yet oftentimes the patient and his family assure you that there has never been any difficulty attributable to the stomach. The usual history is one of severe, continuous epigastric pain with prostration. The onset is severe enough and alarms the patient sufficiently so that medical aid is sought at once. Every one concerned realizes that something very serious has happened. Rigidity of all of the abdominal muscles is absolute from the first. No other condition ever gives rise to such unalterable and unyielding tension of the muscles. Rigidity is a sign common to many acute abdominal lesions, but in no other disease is it so complete, so absolute, so persistently unchanged. This fixed contraction of the muscles in nature's effort to splint the abdomen, embraces the diaphragm, and consequently respiration is short and jerky, with a pronounced expiratory grunt. We have not noted this grunt with any other abdominal lesion. We have seen one patient who had two perforations within a year. On the second occasion the diagnosis was made for us as the patient greeted us with the statement, "Doctor, I have the same thing I had before."

Acute pancreatitis is less common than perforation, and is in reality a comparatively rare disease. In its signs and symptoms it resembles the perforation of an ulcer, and like it, the onset is sudden and characterized by intense epigastric pain. Tenderness and muscle spasm are,

to a large extent, confined to the mid-line above the umbilicus, and the resulting picture is often spoken of as epigastric peritonitis. In this condition there is frequently a deep lividity or cyanosis of the skin, chiefly noted about the face. As has been suggested, the board-like abdomen of ulcer rupture is wanting, and the diaphragm is not involved. Consequently, the irregular breathing and expiratory grunt are not present.

In considering rupture or gangrene of the gall bladder, one soon realizes that there is nothing which is in any way characteristic of the lesion. Possibly one can say in a general way, that the onset of symptoms is more gradual than in any other upper abdominal emergency, and that the patient gets sicker more slowly. One is quite apt to obtain a history of previous attacks of trouble referable to the gall bladder, and it is quite true that the condition is an acute exacerbation of a chronic lesion.

The three conditions which we will consider next, namely, ectopic pregnancy, ovarian cyst with twisted pedicle, and infected or strangulated fibroids, are strictly speaking, gynecological conditions which might justly be excluded from any consideration in a paper on abdominal diagnosis, yet all three may be inaugurated by severe pain with accompanying shock or collapse in varying degrees. To be sure, the pain, tenderness, and muscular involvement are very apt to be confined to the central lower abdomen, but strangulation, either of a cyst or fibroid, may produce rather diffuse abdominal pain with varying degrees of general abdominal tenderness and muscular involvement.

In these pelvic conditions, one usually obtains his greatest amount of diagnostic aid from the history and bi-manual examination. All three of these conditions are accompanied by palpable pathology, although to be sure, two or more factors may unite to frustrate our attempts at palpation. What we have said of ectopic pregnancy, refers to the condition before rupture takes place. After rupture, the picture is one of internal hemorrhage, which, with the other signs and symptoms present, usually allows a very accurate diagnosis.

Our chief difficulty lies in making a diagnosis of ectopic pregnancy before rupture, and the most common pitfall is a chronic inflammatory tube. Between these two conditions, if a man makes a correct diagnosis of ectopic in fifty per cent of his cases, he is doing rather well. One of our cases shows how the diagnosis may be further complicated. The history was very typical and associated with pain on the right side, but bi-manual examination disclosed a mass the size of a hen's egg posteriorly and to the left of the uterus. How were we to account for right sided pain with a left sided mass? Operation disclosed the following condition:—the right tube was the seat of an ectopic pregnancy and

explained the right-sided pain, as the abnormal stimulus was referred along the usual paths. In addition the pregnancy was well out in the tube toward the fimbriated extremity, and the tube was unusually long. The tube curved upon itself and passed behind the uterus so that the mass lay to the left.

Diverticulitis of the sigmoid, mesenteric thrombosis and inflammation of Meckel's diverticulum are uncommon lesions and are mentioned only to make our list complete. In these conditions it is almost impossible to make an accurate diagnosis, and the best one can do is to recognize that a severe abdominal lesion exists and advise immediate laparotomy. The possible exception to the foregoing statement may be in connection with diverticulitis when a palpable mass is present.

Thanks to modern preventive medicine, typhoid fever is fast disappearing from our midst, and the rising generation of internists and surgeons is little interested in typhoid perforations. It is sufficient to remember that such a condition is possible. Fortunately, a typhoid perforation usually occurs during the third week of the disease, a time when the diagnosis of typhoid fever has been firmly established. The only characteristic symptom is sudden, severe pain in the lower right quadrant of the abdomen. The text book picture with its obliteration of liver dullness and high white blood count means that conditions have progressed beyond the point where surgical intervention can be of aid. If we are to accomplish anything in this catastrophe, we must make our diagnosis early.

In considering the question of intestinal obstruction, one is dealing with a subject which calls for at least a paper by itself. Consequently I will not be able to do it justice, but can only refresh your memory concerning certain points which must be borne in mind when considering acute abdominal lesions. Vomiting is usually given an important place in the symptomatology of intestinal obstruction, but as I pointed out earlier in this paper, it is a symptom more or less common to all acute abdominal lesions. There is nothing characteristic of the vomiting in obstruction unless it be fecal, a condition which we should seek to forestall. The pain with obstruction is usually rather vaguely localized and gives us little or no aid. I have previously referred to Dr. Jones' statements with regard to the location of pain in obstruction, statements which we are unable to corroborate. Our one best diagnostic symptom of intestinal obstruction is complete obstipation in a patient who is obviously sick. I say obviously sick for this is oftentimes the deciding factor in making a diagnosis between mechanical obstruction and impacted feces. Naturally, the first thing to do in considering a case of obstruction is to rule out an external hernia,—something which I am

sorry to say is not always done. Also one must consider the possibility of a hernia having been reduced "en bloc." This is an unusual condition but I have seen one case. The patient, a man, had had a right indirect inguinal hernia for a number of years, which had never given him any trouble. The hernia was easily reduced and was well controlled by a truss. On the occasion in question the hernia became strangulated and the patient, being unable to reduce it, called his family physician, who, without undue trauma, reduced the hernia, but the symptoms of obstruction persisted. When I saw the patient, he had a hernia which could apparently be reduced with very little manipulation, and in fact disappeared itself under ether. In view of the fact that obstructive symptoms had persisted, the patient's abdomen was opened, and a condition found which has given the lesion its name. The strangulated portion of the intestine had been pushed into the abdominal cavity, but had separated the peritoneum from the muscles, carrying it before the retreating hernia. The constriction, which was the neck of the sac, still existed.

Having ruled out an external hernia, the next step is to ascertain whether or not there has been any previous abdominal operation. Any operation which has involved intra-abdominal manipulation, no matter how slight, is a potential source of intestinal obstruction. We recently resected two feet of gangrenous intestine due to an obstructive band near the ileocecal valve, resulting from a so-called simple appendectomy performed twelve years previously. In our experience, obstruction from a band caused by a previous operation is a grave condition, as usually the intestine is gangrenous, even in the face of prompt surgical intervention. Having ruled out the more or less obvious causes of intestinal obstruction, there still remain many cases in which we can only say that obstruction exists and operation is imperative. Once within the abdomen, almost anything can be found as a source of the trouble, and a list of the factors which are known to have caused intestinal obstruction is indeed a lengthy affair. In considering a case of obstruction, always remember that it may be the first symptom of an unsuspected carcinoma of the large intestine, and that in some cases this diagnosis may be made with a fair degree of certainty.

In closing I will touch very lightly on a few questions pertaining to acute abdominal lesions, resulting from external trauma, but will exclude penetrating wounds. The abdominal organs may be divided, in a general way, into two classes, namely, solid organs and hollow organs. The first class will include the liver and spleen, while the second class embraces the stomach, urinary bladder, and intestines. It is obvious that blows upon the abdomen will react differently upon these two classes. It is much easier

to injure a solid organ than it is to damage a collapsible one. The stomach, intestines, and urinary bladder, being collapsible organs, are seldom injured by external blows. The liver and spleen, being friable, as well as solid organs, are more easily injured, but traumatic lesions in these organs are by no means common. The liver, spleen, and stomach are extremely well protected by the ribs, and have the yielding diaphragm as a bumper. The urinary bladder also has rather complete bony protection within the pelvis. The intestines, being collapsible, loosely attached, and overlaid by heavy muscles, are well guarded. The only closely attached portions of the intestines, namely, the ascending and descending colons, are deep within the abdomen and separated from the abdominal wall by coils of freely movable intestines. The type of blow which causes injury to the liver and spleen, differs from the type which causes rupture of the intestines. The former is usually from an object which extends its force across the entire upper abdomen, while the latter is from one which exerts its force over a rather limited area. The old simile is a rather apt one, that if a man receives an intra-abdominal lesion from striking forcibly across a plank, he will have an injury to his liver or spleen, while on the other hand, if he strikes forcibly against the end of the plank, he will have a rupture of the intestines. It is seldom that one makes an accurate pre-operative diagnosis of an intra-abdominal injury, but oftentimes he can hazard a very likely guess.

Two points may be mentioned in connection with rupture of the spleen. One is, that the alarming symptoms may be much delayed from the time of injury, and the second is, the noting of "Balance's sign," which may be illustrated as follows. With rupture of the spleen there is profuse intra-abdominal hemorrhage, and the blood settles into the left loin. Percussion will reveal dullness in the left flank with resonance in the right flank. Now if the patient is turned upon his right side, the fluid portion of the blood will gravitate to the right, but the large clots will not, and dullness persists in the left flank. When the patient is next turned upon his left side, the fluid flows back, and the right side is tympanitic.

It is oftentimes difficult to even determine that an intra-abdominal injury exists, for when

two apparently identical cases present themselves, one may have a lesion and the other not. A few years ago we had a patient who, while driving an automobile at a considerable rate of speed, ran into a telegraph pole and was thrown forward, so that the steering wheel came in violent contact with his upper abdomen. When we saw him he was in shock with a rigid, tender abdomen, but without marked signs of hemorrhage. We decided a laparotomy was justified. Remembering what a blow across the upper abdomen indicated, we made a diagnosis of liver involvement. At operation we found the gall bladder torn from its attachment to the liver, and held only by the cystic duct and artery. There was some laceration of the liver where the gall bladder had been attached. Within a year we have had a patient who was involved in a precisely similar accident. He presented exactly the same symptoms as the first case, and in addition he had had a half grain of morphine, which had not relieved him in any way and his pain seemed intense. Remembering the first case, we operated, only to find an absolutely normal abdomen.

I will mention but one case in illustration of intestinal rupture. A man riding a motorcycle at a high rate of speed, ran into the abutment of a dry bridge and was thrown over the handlebars of the machine, striking his abdomen against the end of a post, a severe blow distributed over a small area. Operation disclosed a portion of the small intestines severed completely across, but what impressed us the most was the fact that this wound was not ragged in any respect, but was just as straight and clean as it would have been had a knife been used to produce it.

I can say nothing concerning traumatic rupture of the stomach, as I have never seen a case, although it is not an unknown condition.

Rupture of the urinary bladder is also a very rare injury when occurring alone. It occurs with fracture of the pelvis, but due to an abdominal blow it is uncommon. I saw one case as an intern. The injury was produced by a human kick.

I realize that my consideration of this very interesting subject has been extremely sketchy. If this paper has recalled only one forgotten fact, it has accomplished its purpose.

## SOME CLINICAL ASPECTS OF PRIMARY CARCINOMA OF THE PANCREAS\*

BY B. M. FRIED, M.D.

THE diagnosis of a primary carcinoma of the pancreas, even in the advanced stages of the disease, is difficult; and to differentiate between a pancreatic and hepatic tumor, or between a

tumor and an inflammatory condition of the pancreas is often impossible. "When one takes into consideration," says Friedenwald, "how extremely difficult it may become to differentiate, for instance, between duodenal ulcer and cholecystitis at times, and that even after a thor-

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ough clinical study, including Roentgen-ray examination, the diagnosis can be established only after exploratory incision, it may be readily understood how much more complicated the diagnosis of malignant disease in this area (right upper quadrant) becomes."

The complexity of the diagnosis of a primary pancreatic cancer is to a large extent connected with the topography of the pancreas. It will be remembered that this organ which measures about 15 cm. in length, crosses the entire length of the epigastrium where it comes in contact with almost every structure situated in this area. The head of the pancreas is in contact with the duodenum; the body with the transverse colon, inferior vena cava, renal veins and the aorta. Finally the tail of the pancreas is in close connection with the spleen.

Since cancer of the pancreas may take origin in any part of the organ, head, body or tail, it seems strange that authors in discussing the symptomatology of epithelial malignancy of the pancreas, do not discriminate between these different parts of this organ, but speak in general terms of cancer of the pancreas. From a clinical point of view, the first symptoms of the disease vary greatly in accordance with that part of the organ which is primarily involved by the new growth.

It goes without saying that in the early stages—and how long these are we do not know—cancer of the pancreas, like cancer elsewhere in the body, runs either a silent course or its symptoms are entirely indefinite.

Bard and Pic<sup>2</sup> were the first accurately to describe the clinical symptomatology of primary carcinoma of the head of the pancreas. These authors almost half a century ago formulated a group of "cardinal symptoms" of carcinoma of this particular part of the organ. (1) A deep, progressive, painless jaundice; (2) A distended and palpable gall bladder; (3) Cachexia, which runs a rather rapid course, being accompanied by loss of weight and weakness. The jaundice designated by the Germans as "Swarzsucht" instead of "Gelbsucht" because of its blackish hue, is sudden in onset and without remissions. It is due to compression of the bile ducts by the tumified head of the pancreas. It is accompanied by a choloria, pruritis and bradycardia.

As seen, the symptoms peculiar to carcinoma of the head of the pancreas are due to the topography of the organ; that is to say, the head of the pancreas, enlarged by tumor, presses upon neighboring structures (the common bile duct), leading to the characteristic jaundice and enlarged gall bladder.

Cancer of the body of the pancreas is relatively less frequent than that of the head. Curiously enough, from the standpoint of symptomatology, it appears to be an altogether different disease. Whereas cancer of the head is characterized by a peculiar icterus and a com-

plete lack of pain, that of the body is as a rule accompanied by the most severe and excruciating pains in the absence of icterus. Chauffard<sup>3</sup> was the first to note this distinguishing feature of primary carcinoma of the body of the pancreas, which he says is one of the most painful abdominal diseases. At the onset of the disease the pain which is usually confined to the epigastrium, comes on in paroxysmal attacks at various intervals. Later the pain is more constant. It is sharp, not related to meals, frequently girdle in character, resembling the "crises gastriques" in tabes, and radiating to the spine or the thorax. There is often a diarrhoea accompanied by a colicky pain and tenderness but no jaundice.

In the case to be reported which the author had an opportunity to study, the tail and the body of the pancreas were both involved by the newgrowth, which lead to the characteristic Chauffard's syndrome.

#### REPORT OF CASE

*A history of excruciating epigastric pain of one year's duration in a man of 70. Death. Necropsy finding: Primary carcinoma of tail and body of pancreas with metastases to lungs and liver.*

*History:*—The patient was a married man of about 70. His chief complaint was pain in the epigastrium, cough and progressive weakness. His family and past histories were unimportant.

*Present illness:*—Began in the spring (April or May) 1925, when the patient noticed that he was getting progressively weaker and also that he was losing flesh. Two-three months later he began to complain of a sharp pain in the epigastrium a little above the midline between the umbilicus and the xyphoid process. The pain was sharp, distressing, was not related to meals or micturition and occurred at irregular intervals, during day or night time. In July 1925 the attacks of epigastric pain increased in frequency and intensity. They used to literally "double" up the patient. They were apparently worse on lying down, and radiated to the chest. He has never vomited, but noticed a diarrhoea from 5 to 6 times daily and also distressing tenesmus.

*Physical Examination:*—This showed an old man with evidences of cachexia and a considerable loss of weight. There was no jaundice or pruritus. The thoracic examination apart from an emphysema was negative. The blood pressure was 115/60.

The abdomen was scaphoid. There was a palpable mass the size of a small lemon in the left upper quadrant in the region of the spleen. This seemed to be slightly irregular, somewhat hard and immobile.

The clinical pathology was negative.

*Course:*—Four or five months before the end the patient suffered greatly from frequent attacks of the sharp excruciating epigastric pain described above. His diarrhoea and tenesmus grew worse daily. He lost ground rapidly and died in an advanced stage of cachexia with emaciation.

*Necropsy:*—This was performed 6 hours after death. The peritoneal and pleural cavities showed no free fluid. The heart was small, weighing 250 gm. The lungs showed chronic adhesive pleuritis, emphysema and a terminal broncho-pneumonia.

On the anterior border of the right lung corresponding to the junction of the sternum with the 4th rib a small tumor nodule was found.

The stomach and intestines showed nothing of importance. Feces of a normal color were found in the

lower part of the large bowel. The liver was very small, weighing only 900 gm. Beneath the capsule of Glisson there were a few small tumorous nodules 1 cm. in diameter.

Histologically the liver showed pronounced atrophic changes.

The gall bladder and the ducts were normal.

**Pancreas:**—When the stomach was removed a mass was disclosed in the left subcostal region, strongly adherent to the spleen. The latter was small, being surrounded by a thick fibrous capsule. The lower segment of the tumor covered the left adrenal and also the upper part of the left kidney to which it was also strongly adherent.

Examination showed that the tumor mass occupied the entire caudal part of the pancreas and also a large part of the body. The neck was normal. The head of the pancreas was not enlarged, was normal in color and consistency, and had its usual duodenal attachment.

at different angles forming tubules resembling in size and arrangement the normal pancreatic acini (Figure 2) or in diffuse masses (Figure 3). These latter are separated from each other by thin fibrous partitions approaching those seen normally between the pancreatic lobules. Mitotic figures are not uncommon. Tumor is found invading the lymphatics. The stroma of the growth is similar to that of the normal pancreas.

At the edge of the tumor there is a very marked lymphocytic infiltration. The islands of Langerhans are hyperplastic in areas invaded by tumor as well as in areas free from the newgrowth. The ducts show no changes of any kind.

It is judged that the tumor originated from the epithelial cells forming the acini.

In this case, then, the tumor which to all appearances has originally started in the caudal part of the pancreas has progressed toward the



FIGURE 1. Primary Carcinoma of the tail of the pancreas. Tumor replacing the tail and the body of the organ.

**Tumor:**—The tumor (Figure 1) with the attached parts of the normal pancreas weighed 300 gm. (The weight of a normal pancreas in an adult is from 100 to 120 gm.) It measured 6 x 9 x 7 cm., being almost stony hard. Its surface was irregular as if it were composed of innumerable small nodules enclosed within a thin translucent capsule.

On cut surface it had a mosaic appearance, being composed of numerous lobules separated by thin strands of fibrous tissue; in gross the architecture and color of the tumor approached roughly that of the normal pancreas.

The duodenal orifice of the duct of Wirsung was patent and a probe could pass for about 5 cm. in the duct. The remote part of the duct was obliterated. The tumor compressed the celiac axis, the solar plexus, and overlay the anterior surface of the aorta without leading, however, to any vascular constriction.

Histologically the tumor is made up of polygonal epithelial cells the size of a large lymphocyte. The cellular cytoplasm is abundant and is finely granular; the vesicular nucleus is large, and is provided with an eccentrically situated irregularly round nucleolus. Individual cells are separated from each other by a narrow clear space.

The cells are arranged either in small groups cut

body of the organ, leading to the excruciating epigastric pain. Here, again, as in primary cancer of the head of the pancreas, the outstanding symptomatology was not due to involvement of the organ proper but was "borrowed," so to say, from its topography. In cancer of the head of the pancreas the dominating factor is pressure upon the ducts resulting in icterus and enlargement of the gall bladder; in cancer of the body the irritation or possibly the involvement by the newgrowth of the solar plexus causing excruciating abdominal pain is the outstanding symptom in the clinical picture.

Abdominal pain in primary carcinoma of the pancreas has been noted by various clinicians. Osler<sup>4</sup> considers "pain in the epigastrium among the most important features in the symptomatology of the pancreas." This keen clinician did not discriminate, however, between the involvement of one or another part of the organ. Joachim<sup>5</sup> in Tice's Practice of Medicine, notes

also that "pain is usually a constant symptom." Barron<sup>6</sup> in an investigation of 29 cases of primary carcinoma of the pancreas says: "Pain is very common." In a recent article Graves<sup>7</sup> discussing the symptomatology of malignant neoplasms of the pancreas outlines that in primary carcinoma of the pancreas "we must rely on

they never die suddenly during the crisis of the 'drame pancréatique'." And later he says: "Of all the cases of cancer of the head of the pancreas, of the ampulla of Vater, and of pancreatic stones which I was able to collect, I found only one in which the 'drame pancréatique' was present. In this case, however, it concerned an infectious lithiasis of the bile ducts." "Such are the facts," continues the brilliant French clinician, "I guard myself from proposing any theories. I state only." A few years later another talented French clinician, Chauffard, reasonably explained "the facts."

Among American clinicians, only Archibald<sup>8</sup> distinguishes carcinoma of the pancreas according to the region involved: 1. The head, with the characteristic jaundice, etc.; 2. the body with the severe epigastric pain and lack of jaundice.

In a recent monograph Gross and Guleke<sup>9</sup> mention also the excruciating pain as typical of carcinoma of the body of the pancreas. They designate it as "le drame pancréatique," which was thought by Dieulafoy to be present in acute pancreatitis only.



FIGURE 2. Primary Carcinoma of the tail of the pancreas. Showing the general architecture of the new growth.

pain. It is commonly epigastric and dull, though I have seen one case in which pain was persistent and intense and was referred to the middle of the back and around the left costal margin. The pain of carcinoma of the pancreas occurred suddenly while the patient was on a shopping tour, and never disappeared. Loss of weight began at once, while there were no gastric or intestinal symptoms of consequence, jaundice did not appear for two months and was never as deep as usual but persisted until death. Exploration disclosed a carcinoma of the size of a hen's egg in the body of the pancreas adjacent to the head."

As indicated, constant pain is noted by all writers as being characteristic of carcinoma of the pancreas in general. As a rule it is rather a gnawing, dull pain or distress characteristic of diseases of abdominal organs in general. In rare instances it is sharp and severe. It is probable that in the advanced stages of the disease, when the patients oftentimes present themselves at the clinic, the tumor has spread toward the body of the pancreas, leading to a characteristic agonizing pain.

Dieulafoy<sup>6</sup>, in his "Manuel de Pathologie Interne," gave an admirable description of the "douleurs atroces a la région ombilicale, a l'épigastre, aux hypocondres" in acute pancreatitis which he has designated as "*le drame pancréatique*." He is, however, surprised that the "drame pancréatique" is never observed in carcinoma of the head of the pancreas. "The patients" (with cancer of the head of the pancreas), he says, "succumb to their cancer, but



FIGURE 3. Primary Carcinoma of the tail of the pancreas. A detail (X850) showing one "nest" of cells and the type of cells—large epithelial cells having a large nucleus with a centrally located voluminous nucleolus.

Numerous explanations have been evolved for this remarkable pain. By some it is believed to be due to pancreatic perineuritis; by others to stasis of the pancreatic secretion. The most plausible explanation, however, is given by Chauffard<sup>3</sup>, who attributes the pain to pressure and possibly to involvement by the tumor of the solar plexus.

Thus to the "syndrome pancréatico-biliare," an expression coined by Dieulafoy to outline the features characteristic of cancer of the head of the pancreas, Chauffard adds what he calls the "syndrome pancréatico-solaire" pathognomonic of carcinoma of the body of the pancreas.

## SUMMARY

In the early stages primary carcinoma of the pancreas, like carcinoma elsewhere in the body, runs a silent course.

The first symptoms of a primary cancer of the pancreas depends from the part of the organ primarily involved by the newgrowth: In cancer of the head of the pancreas the symptoms are due to compression by the tumor of the bile ducts, leading to a peculiar jaundice and as a rule a distended gall bladder.

In instances where the primary seat of the tumor is the body of the pancreas the patients' chief complaint is a sharp excruciating epigastric pain probably due to pressure or involvement by the tumor of the solar plexus. Jaundice is lacking.

Severe pain may also be present in cancer of

the head or tail of the pancreas in instances when due to a protracted course, the newgrowth from these parts of the organ has probably reached the body and has invaded the solar plexus.

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## A PLEA FOR CONSERVATISM IN THE TREATMENT OF OPTIC NERVE DISTURBANCES FROM FOCAL INFECTION\*

BY LEON E. WHITE, M.D.

NEEDLESS, or too radical, intranasal surgery in cases of optic nerve involvement from focal infection is the subject presented for your consideration. The data are derived from my last sixty unpublished cases. Some of these were seen only once in consultation, but as the results are known they have been included in my series. In my absence several were followed by my son. The few that failed to carry out my recommendations are incomplete.

I hope to show from this study that it is usually possible to find the focus of infection and by eliminating it before the nerve is damaged, to obtain prompt recovery. It will be shown that in the majority of the cases only the focus needs to be eliminated, although it is advisable to remove nasal obstruction when well marked. In but few cases has it been necessary to open the posterior sinuses, and even in some of these it was needless or done too late. The routine opening of uninfected sinuses has been found unnecessary and is to be condemned.

The papers constantly appearing on this subject, I regret to say, nearly all advocate too radical intranasal surgery. While some writers merely recommend a unilateral ethmoid and sphenoid exenteration, others are only satisfied with a bilateral one, to which in some instances is added a tonsillectomy. I have been expecting that in addition to the bilateral exenteration of the sinuses and the removal of the tonsils, some one would advocate extraction of all the teeth. This would seem about as sane as the wholesale exenteration of uninfected sinuses. Teeth that often excel in beauty and utility might replace those extracted, but as yet nothing has been devised to replace the sinuses.

\*Read before the Philadelphia Laryngological Association on November 2, 1926, by invitation.

Before becoming too critical let me admit that in my early studies sinuses adjacent to the optic nerve seemed the all important foci of infection and that by draining them the cases would be cured. My first seventeen cases were attributed to the sinuses. Most of the papers appearing at the present time still maintain this view. This is largely due to the fact that the opening of the accessory sinuses is done about the time the case would normally commence to improve, or to the benefit following the depletion of the structures adjacent to the nerve. Teeth or tonsils are rarely mentioned.

Volumes were written on the anatomic relationships of the optic nerve to the posterior sinuses, and while I should hesitate to say that none of this study was valuable, for much of it was enlightening, still I do maintain that it threw sinuses too much into the limelight while the more remote sources of infection were decidedly left in the background.

In my effort to find sufficient pathology in the sinuses to account for the optic nerve disturbances, I had careful examinations made of the tissues removed from the sinuses and turbinates. While this tissue frequently showed changes, it failed to convince the pathologists that it was sufficient to account for the eye conditions. More remote sources of infections were then sought and soon tonsils presented a focus which loomed up as an important etiologic factor. From tonsils it was but a step to teeth, so I began to have the teeth filmed in all cases. Along with the teeth and tonsils the antrum took on added significance, not as a sinus from which there might be direct extension to the optic nerve, but as a focus from which infection might pass by way of the blood stream or lymphatics to the



nerve. At first it was the antrum with pus that was especially sought, but later marked thickening of the mucosa seemed fully as important.

At present, as I look in retrospect over more than one hundred cases, it seems that the infection comes either from some systemic condition, as influenza or intestinal toxemia, or from some definite focus such as teeth, tonsils or sinuses; that the infection travels by way of the blood stream and produces a neuritis of the optic nerve the same as of any other nerve. Eliminate the infection from the blood stream and the neuritis subsides. The danger lies in the fact that as the optic nerve is a highly specialized structure, it degenerates easily and never regenerates. There is always a possibility that in a severe neuritis optic atrophy may intervene before the inflammation subsides. To this end let us consider the type of case where this might happen and the methods of combating it. It is a well recognized fact that the majority of the cases of acute optic neuritis recover spontaneously so that if there were some method of distinguishing between those that might reasonably be expected to recover from those that might not, they could at once be dismissed.

As to the methods of differentiating the cases, the duration and extent of the loss of vision, the condition of the fundus, the virulence of the infection and the size of the optic canals must all be considered.

*Duration:* The conclusions reached from an analysis of the cases were that unless there was improvement under treatment within a week there was danger of permanent impairment of vision, while in cases of more than two months' standing little could be expected except possibly checking the loss of vision by the removal of some definite focus of infection.

*The Amount of Vision:* It was also found that the demand for early operative interferences in total loss of vision was more imperative than when the loss was but partial.

*Condition of the Fundus:* When the nerve appears normal one might, with safety, delay operating much longer than when there is increasing engorgement or commencing pallor.

*The Virulence of the Infection:* As in all the other types of infection, so in that producing optic neuritis, the microorganisms differ greatly in virulence. When the infection is of the virulent type there is probably considerable exudate about the nerve or even within its sheath. The optic nerve, as you know, is really not a nerve but a part of the brain. It is easily destroyed and does not regenerate. Parsons says of it: "The so-called optic nerve, together with certain parts of the retina, constitutes a lobe of the brain, and has, therefore, the characteristics of the central nervous system. Hence the nerve fibers are devoid of a sheath of Schwann and the interstitial substance is neuroglia."

*The Size of the Optic Canal:* That the osseous optic canals normally afford great protection to

their neural contents cannot be doubted, but when these canals become narrowed and the enveloping sheath of the nerve, or the nerve itself, swollen the unyielding walls may cause constriction sufficient to produce atrophy.

Many ophthalmologists have maintained that most cases would have recovered if nothing had been done and have intimated that the brilliant results following intranasal surgery would have taken place had nature been permitted to work unhampered by the enthusiastic rhinologist. The fact remains, however, that blindness does follow in a certain number of these cases. The problem is to separate the cases where spontaneous recovery might be expected from those where permanent damage would follow if untreated. Rhinologists have been criticized for operating on all cases irrespective of their severity. They justified these procedures by claiming that by these methods they would relieve those cases where blindness might follow if untreated and maintained that these operations were without danger. It soon became apparent, however, that bilateral extirpation of the ethmoids, for instance, was not devoid of danger.

Cushing, having seen several cases of meningitis following ethmoid extirpations, did us all a valuable service in calling attention to these fatal results and halted somewhat the tendency for too radical and too irrational intranasal surgery. The word "irrational" refers to the numerous cases he reports where intranasal surgery was performed for the relief of blindness due to brain tumors, a sad commentary on the lack of diagnostic skill on the part of the rhinologists.

To return, however, to the original discussion, —Is it possible after a careful study of these cases to differentiate between them? To a considerable extent, yes. To an absolute certainty, no. There are cases so slight and transitory that it would be little short of criminal to submit them to any surgical procedure not indicated in our ordinary rhinological findings. Infected teeth or tonsils should be removed. If the antrum shows infection it should receive appropriate attention. But to extirpate ethmoids and sphenoids not so evidently diseased that they require opening irrespective of the eye condition, is, I believe, a gross but too common error. Mild cases recover without treatment or with ordinary astringent sprays. These cases are usually taken too seriously. Extreme measures are instituted where little or no treatment would suffice, and while recovery may take place in spite of, rather than because of, these procedures, the patient is submitted to needless risks and the intranasal structures are unnecessarily mutilated. Then again there are cases with marked loss of vision yet with a normal optic canal. Here again the focus should be diligently sought and, where possible, removed. Should the posterior sinuses be markedly obstructed by an enlarged middle turbinate or a deflected septum, aeration of this region is demanded by the re-

moval of the obstruction. In the ordinary run of cases reliance should be placed on hot alkaline douches, astringent sprays, suction, etc. These first two classes probably comprise 85% of all the cases arising from focal infection and recovery in all should be obtained by conservative methods. In the remaining 15% where we are contending with a severe neuritis with marked or complete loss of vision, especially in a patient with an optic canal considerably below normal, I believe that in addition to the removal of the focus it is usually advisable to also remove the middle turbinate, take down the front wall of the sphenoid and uncap the posterior ethmoid cell. This procedure is especially advocated if the blindness is of several days' duration and the fundus shows increasing engorgement or commencing atrophy. In none of the cases do I advocate exenteration of any of the other sinuses unless they show pathology sufficient to warrant it, irrespective of the eye condition. Since I have followed the procedures above indicated my results have been as good as when, as a matter of routine, I opened the posterior sinuses.

So much has been said and written on the intimate relationships between the optic nerve and the sinuses that it has become almost an obsession with rhinologists to consider the sinuses, even if normal, in some mysterious way responsible for the eye disturbances. An explanation of this mystery was attempted in a paper I presented before the American Laryngological Association in Montreal in June, 1926, entitled "The Influence of Negative Pressure in the Sphenoid on the Optic Nerve." In this article it was explained that the benefits usually following the opening of the posterior sinuses resulted from the relief of the negative pressure and not from the removal of a focus of infection.

Onodi's writings on the contra-lateral relationship of the optic nerve to the posterior sinuses has undoubtedly led to the needless bilateral exenteration of many normal sinuses.

At the Dallas meeting of the American Medical Association in 1926 several extremely interesting papers on iritis were read before the Ophthalmological Section. A paper by Iron and Brown entitled "Recurrence of Iritis as Influenced by the Removal of Infections" deals with fifty cases of iritis followed from three to twelve years. The probable source of the infection was found in forty-six of these cases. The teeth alone were considered the focus in 20%; the tonsils in 34%; the sinuses (antrum) in 2%; the teeth and tonsils combined in 10%; the sinuses, teeth and tonsils in 4%; and the sinuses, teeth and colitis in 2%. Thus, it would seem that sinuses play a minor role in iritis as they do in optic neuritis.

Zentmayer in a paper entitled "The Prostrate as a Remote Focus of Infection in Ocular Inflammations" directed attention to the impor-

tance of searching further than the mouth, nose and throat for the portal of infection. He states that it is not infrequently found in the prostate gland.

A third paper by Mills entitled "Ocular Disease Occurring in the Course of Nondysenteric Amebiasis" calls attention to the importance of investigating the intestinal tract. He states that "it is certain that the arrest or cure of chronic iridocyclitis, choroiditis, retinitis, episcleritis, keratitis and conjunctivitis has followed the faithful use of ipecac and its derivatives when the recognition of a protozoan or flagellate background has ended repeated vain searches for a bacterial etiology." He furthermore states that in his sole case of double optic atrophy three varieties of endamebas were found at the first examination of the stools, "and that all other tests including roentgenograms of the brain, sella and sinuses were without results."

It may interest you to know that Dr. George Derby's treatment for retrobulbar neuritis when no definite focus is found is to put the patient in bed, sweat him, flush out the kidneys and intestinal tract. That the appendix and gall bladder may be sources of infection to the blood stream is now well recognized. One of my cases of retrobulbar neuritis was attributed to infection in the pelvis.

It is imperative that one who studies optic nerve lesions should be conversant with all possible sources of infection. Owing to their proximity the sinuses have long shouldered the responsibility for most of these disturbances. This burden must now be distributed between the teeth, tonsils, sinuses, prostate, appendix, gall bladder, fallopian tubes and intestinal tract.

You may be interested in our method of investigating cases: Patients are always referred from either an ophthalmologist or from an ophthalmological clinic, so that the eye report usually accompanies the patient. This should give a history of the case, its duration, vision, visual fields when possible, and condition of the fundus. A correct diagnosis is naturally essential before surgical intervention is advised. The symptoms in the ordinary cases of acute optic or retrobulbar neuritis are quite typical.

Associated with the loss of vision there may be discomfort about the eye or lameness on moving it. Occasionally there is slight exophthalmos, this condition indicating an inflammatory process in the orbit. Pupillary changes and ptosis are found occasionally. There is frequently nyctalopia, a central scotoma for colors, enlargement of the blind spot and contraction of the fields. The patients generally say that things at first were blurry when looking straight ahead, but comparatively clear when viewed sideways. Changes in the fundus are of great diagnostic value, varying, as they do, from the normal to a commencing pallor in some cases, and in others to a marked redness and swelling about the

nerve, with engorgement and tortuosity of the retinal veins. It is essential that these inflammatory swellings be differentiated from the edema due to intracranial pressure.

A careful examination is made of the nose, throat and mouth, the sinuses transilluminated, and the position of the septum and size of the middle turbinates are especially noted. The anterior portion of the nose frequently appears normal. The one vital point to determine, in the nasal examination, is the size and position of the middle and superior turbinates. Do they block the ventilation of the posterior sinuses? Is there impaired aeration? It takes but a slight obstruction to interfere with the ventilation, and the mistake is frequently made of expecting to discover marked changes; while, on the other hand, whenever there is no blocking of the normal openings of the posterior sinuses, this region can, I believe, be ruled out as a causative factor in the neuritis. The following is quoted from Stark, who expresses well the thought I wish to convey: "From a nasal standpoint we must not expect to find the common symptoms of sinus infection, pus, polypus, history of nasal discharge, etc., as we are dealing with a closed sinus; otherwise we should not have pressure. . . . The deflected septum and middle turbinate tightly pressed against the lateral wall should always be suspected."

It is not always possible to determine the health or disease of a tonsil. Special attention is given to any enlargement of the cervical glands below the angle of the jaw. Congestion or swelling about the soft palate and pillars usually indicates infection. The tonsil is usually probed and pulled into the throat to determine its size and general appearance.

The teeth come next and it is right here that snags are usually struck. The patient as a rule has a good dentist and resents fault being found with the teeth. The patient's dentist was formerly asked to look the teeth over for possible infection but these reports were usually valueless. Dentists as a class try to preserve many teeth that undoubtedly need extraction. Bridge work, capped and pivot teeth are especially liable to harbor infection. If the teeth are suspicious or if no other probable focus has been found, an expert odontologist is asked to look them over. He films them, notes the fillings and reports fully on every tooth that is at all suspicious, devitalized ones especially. By this careful method of investigation the teeth are now found to be one of the most common foci of infection. Dr. Rice has kindly consented to discuss this phase of the subject. The paper he presented before the Triological Society in Montreal was a masterpiece. It stated the subject of focal infection from teeth in exactly the way I would like to have every ophthalmologist and rhinologist view it.

Now as to the sinuses, the patient is referred to a radiologist who usually takes two antero-

posterior and one lateral plate of the sinuses and one of the optic canals. Should the pituitary be under suspicion additional plates may be needed of the sella, but as a rule this region is well shown on the one lateral plate. When even the sinuses are negative the teeth and tonsils should be suspected. Tonsils at all suspicious, with teeth and sinuses negative, should be removed. The diagnosis many times must be made largely by exclusion. In the hospital cases physical and neurological examinations are made a part of the routine as well as a Wasserman. In private practice this is done in all doubtful cases. One should consider in turn blood, urine, hysteria, pellagra, lues, tobacco, alcohol, lead, arsenic, quinine, etc. Pituitary disease, brain tumor and multiple sclerosis, while not usually producing such sudden loss of vision, must always be borne in mind. As a matter of hospital routine the various cranial nerves and lobes of the brain are tested. Not a few cases with brain tumor have had various nasal operations before they were correctly diagnosed. It is usually possible to accumulate all this data within forty-eight hours. It is always best to look at these cases in a quiet, sane manner. The patients are usually greatly disturbed over their loss of vision, so let us not become unduly excited as most of them would recover if left alone. Should some definite focus be found, this should be eliminated unless the patient is already much below par. Should he be convalescing from influenza or have some other debilitating disease, there might be danger of losing his life. Three or four of my patients were such poor surgical risks that it was necessary to wait some weeks. Unless some definite focus is found, it is usually best to treat the case. The intestinal tract should be kept healthy and the patient put in the best possible condition to combat his infection, as in many instances retrobulbar neuritis only comes when his resistance has been lowered.

In some cases the drainage and ventilation of the posterior sinuses is impaired possibly by negative pressure. This would favor the migration of bacteria and toxins from the blood stream to the optic nerve, a condition similar to what usually exists in the tympanum in Bell's palsy. In such cases it has been my practice to use a nasal spray of 10% argyrol and adrenalin about the middle turbinate every three or four hours. Hot saline irrigation, tampons of argyrol and hot mineral oils all tend to assist in the establishment of the ventilation of the sinuses. In about 15% of the cases where there is reason to fear optic atrophy, removal of the middle turbinate and opening of the sphenoid and posterior ethmoid cell is advocated.

I have in my records sixty unreported cases with some type of optic nerve disturbances. The detailed report of thirty-three of these appeared in the *Annals of Otolaryngology and Rhinology*, December, 1926. These cases will, I believe, substantiate my contention that most cases

recover by simply removing the focus of infection.

The tonsils appeared to be the only focus in seventeen, the teeth in fourteen and the antrum in two. Both teeth and tonsils appeared infected in four cases, while in six it was found in teeth, tonsils and antrum. In but six cases were the ethmoids involved and even in these there was also infected teeth, tonsils or antrum.

The posterior sinuses were opened in but nine of these cases. In two recovery would probably have taken place fully as speedily had the sphenoids not been opened, while in five it did not benefit. Thus, in only two out of the sixty cases was it of any special value.

A definite focus was found in fifty-three while in three it was thought to be systemic, diabetes in one, hyperthyroidism in one, post-scarlatinal in one. Two cases were negative except for marked blocking by hypertrophied middle turbinates. A focus was not discovered in one case, while another had an acoustic neuroma.

The surgical elimination of the focus was followed by normal vision in twenty-three, marked improvement in twelve, slight in five, none in six. In seven refusal to have it removed was followed by no improvement in six and normal vision in one. Three cases recovered under treatment, while one showed marked improvement. A fifth case (the diabetic) did not improve. The patient with the acoustic neuroma died as did one with carcinoma of the sphenoid.

Thus, teeth and tonsils appeared to be the foci in about 70% of these cases while the ethmoids were involved in but 10%, so there would seem little excuse for longer considering ethmoids and sphenoids the all important factors in optic nerve disturbances. They rarely require exenteration. The important thing today in rhinological surgery, as in all other branches, is to remove the focus.

#### SUMMARY

It is usually possible to find the focus of infection and by eliminating it, before the nerve is damaged, to obtain prompt recovery. Nasal obstruction when well marked should be removed. As the posterior sinuses are rarely infected it is unnecessary to open them except in a few desperate cases. The advocacy of exenteration of the ethmoids and sphenoids, even when they show no definite infection, is to be condemned. The benefit following the opening of the sinuses would naturally occur about that time, although some of it might be due to the depletion of the structures adjacent to the nerve. As sufficient pathology was not found in the sinuses to account for these optic nerve disturbances more distant foci were sought. The removal of infected teeth and tonsils soon proved to be the most effective method of treatment. Infection is now believed to come from either some systemic condition as influenza or intestinal tox-

emia or from some definite focus in teeth, tonsils or sinuses. This infection travels by way of the blood stream and produces a neuritis of the optic nerve. Eliminate it from the blood stream and the neuritis subsides.

The prognosis is dependent upon the duration and extent of the loss of vision, the condition of the fundus, the virulence of the infection and the size of the optic canals.

About 85% of the cases would recover under conservative treatment such as local medication and the removal of some definite focus. These cases are usually taken too seriously. Extreme measures are instituted where little or no treatment would suffice, and while recovery may take place in spite of, rather than because of, radical operative procedures, the patient is submitted to a needless risk.

In the remaining 15% where we are contending with a severe neuritis with marked or complete loss of vision, it is usually advisable, in addition to the removal of the focus, to also remove the middle turbinate and open the posterior sinuses.

Teeth should be studied by an expert odontologist, devitalized ones especially.

Negative pressure in the sphenoid would favor the migration of bacteria and toxins from the blood stream to the optic nerve, a condition similar to that which takes place in the tympanum in Bell's palsy.

A definite focus was found in fifty-three of the sixty unreported cases. The tonsils appeared to be the only focus in seventeen, the teeth in fourteen and the antrum in two. Teeth and tonsils either alone or combined was considered to be the focus in 70%. In but six cases were the ethmoids involved and even in these there was also infected teeth, tonsils or antrum. The frontal and sphenoid were each involved but twice. The removal of the focus was followed by normal vision in 50% and marked improvement in 25%. Much better results would have been obtained had the focus been removed earlier.

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## THE USE OF DIETS WITH VERY LOW PROTEIN CONTENT IN THE TREATMENT OF CHRONIC NEPHRITIS WITH NITROGEN RETENTION

BY MILLARD SMITH, M.D.\*

It is generally conceded that restriction of protein in the diet is desirable in the treatment of advanced nephritis with nitrogen retention. The reason for giving diets of low protein content is to prevent an increase in the amount of nitrogen retained and to reduce as much as possible the existing retention. At present diets containing 1800 to 2000 calories and 20 to 40 grams of protein are given somewhat arbitrarily, the amount of the protein being fixed by general clinical considerations. Instead of depending upon a clinical estimation of the degree of renal insufficiency in order to fix the amount of protein in the diet, it would be desirable to have some fairly accurate criterion from which the proper amount of protein might be calculated.

The functional capacity of the normal kidneys, when considered in terms of the excretory work which can be performed during twenty-four hours, exceeds considerably the usual amount of work thrown upon them. With renal damage occurring in chronic nephritis the functional capacity may be so lowered that in twenty-four hours all of the waste products from the usual food intake and the body catabolism cannot be excreted; one result is nitrogen retention. This is evidenced by an increase of the blood non-protein nitrogen. In order to prevent this retention it is necessary that the total nitrogen catabolized by the body should not exceed the functional capacity of the kidneys, and, if it is desired to relieve the retention, the nitrogen catabolism must be less than the twenty-four hour excretory capacity of the kidneys. The functional, or excretory, capacity of the kidneys may be determined by an analysis of non-protein nitrogen in the twenty-four hour urine. Just as the amount of water which will run out of a given orifice depends upon the head of pressure, so the amount of nitrogen excreted by the kidney, having a given maximum functional capacity with other things being equal, will depend upon the height of the non-protein nitrogen in the blood. So with renal insufficiency and nitrogen retention the twenty-four hour nitrogen excretion will decrease as the blood non-protein nitrogen becomes lower.

The nitrogen which is excreted comes from two sources, endogenous and exogenous. The exogenous nitrogen is that taken with the food in the form of protein. It is approximately accurate to state that all of this nitrogen is presented to the kidneys within twenty-four hours for excretion. The endogenous nitrogen comes from the breakdown of body tissues and, in the

normal subject, is only a small part of the total nitrogen presented for excretion, providing the body is not satisfying some of its caloric requirements with tissue protein.

It is quite simple to estimate the amount of protein which should be allowed a given patient with nitrogen retention in order that the retention may be relieved. The non-protein nitrogen should be determined in a twenty-four hour specimen of urine, preferably for two or three days to get an average, as it will vary somewhat from day to day. The protein intake should be adjusted so that the nitrogen resulting therefrom (grams of protein  $\div$  6.25 = grams of nitrogen) is less than the non-protein nitrogen excreted in the urine over twenty-four hours. The more the nitrogen content of the diet is reduced below the capacity of the kidneys to excrete nitrogen, the more rapid will be the loss of retained nitrogen from the body.

It is not uncommon to find patients whose twenty-four hour renal functional capacity for nitrogen excretion is so small that it equals their endogenous nitrogen catabolism. As it is not feasible to lower the endogenous nitrogen catabolism indefinitely, it will be evident that these cases are beyond dietary measures of control. In order to lower the blood non-protein nitrogen of others it may be necessary to use protein intakes well below the existing protein requirements. Several questions present themselves in connection with what has been said. What is the minimum protein requirement? What is the minimum endogenous nitrogen metabolism? What is the harm to be expected from not supplying the minimum protein requirement? Will the endogenous nitrogen metabolism increase when the minimum protein requirement is not supplied?

### MINIMUM PROTEIN REQUIREMENT

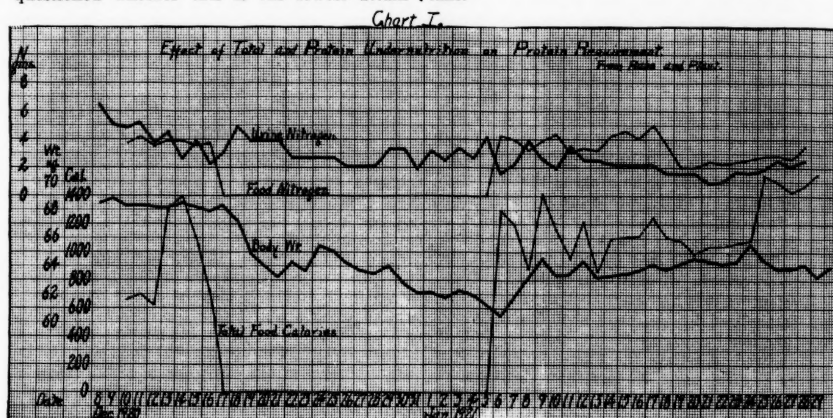
The minimum protein requirement is the amount of protein which will replace the daily loss of this substance from the body. This loss is in the form of urine, stool and sweat nitrogen compounds. There are many technical obstacles which prevent direct determination of the amount of this loss, and it has been the custom to consider the amount of protein in the diet which will maintain an individual in nitrogen balance as the minimum amount. This indirect method actually prevents the finding of the absolute minimum protein requirement for, as it is usually employed, the subject is never allowed to maintain a negative nitrogen balance for more than a few days. Strictly speaking, there is no such thing as an absolute minimum, for as the

\*From the Thorndike Memorial Laboratory, Boston City Hospital.

body protein stores become depleted it requires less and less protein in the diet to maintain the metabolism of the remainder. It is far more important to answer the question of how much body protein is necessary to maintain a subject in activity and health than to determine indirectly the so-called dietary minimum protein requirement. The true minimum protein requirement would be the amount of protein in the diet necessary to maintain the minimum amount of body protein which is indispensable for growth, health and normal activity. As the latter is not known, it follows that the minimum protein requirement is not known. However, the commonly accepted minimum is 0.6 grams of protein per kilogram of body weight. The authority for this standard originated in the studies of Chittenden and Sherman, but it may be questioned whether this is the lowest attain-

ance. However, it demonstrates that 0.63 grams of protein per kilogram of body weight will keep an individual in a healthy state and in nitrogen balance.

Sherman<sup>2</sup> has analyzed the data of twenty investigations concerning protein requirement for normal adults and found that the average intake in grams per kilogram of body weight was 0.7. His examination of the factors determining the amount of protein needed to maintain nitrogen balance showed that "the wide differences in amounts of protein catabolized in the different experiments cannot be attributed primarily to the kind of protein consumed nor to the use of diets of fuel values widely different from the energy requirements. Apparently the most influential factor was the extent to which the subject had become accustomed to a low protein intake."



able minimum, and it is of interest to review their work.

The most extensive investigation into the problem of normal minimum protein requirements is that done by Chittenden<sup>1</sup>. He chose as subjects normal young men from the Army and from the athletic department of Yale University. It is significant that these subjects were allowed to choose the energy content of their diets and no attempt was made to supply this factor of the diet in excess. The total calories supplied never reached 3000 in any of the subjects. Despite this fact the men maintained themselves in nitrogen balance and led an active life, either sustaining or improving their gymnastic records, while their protein intake varied between 0.63 and 0.75 grams per kilogram of body weight per day. As will appear later, this series of experiments distinctly does not represent an attempt to determine the absolute minimum amount of protein on which these men could be maintained while in nitrogen bal-

In the experiments referred to above certain well recognized principles, by means of which it is possible to lower protein catabolism, were not utilized, and, for this reason, it is not correct to infer that 0.6 grams of protein per kilogram of body weight is necessarily the lowest protein intake which will satisfy the body needs.

The most important factors concerned in reducing the protein requirements of the body to a minimum are, (a) the supply of an excess of energy in the form of carbohydrate and fat, so that the body protein will not be used for purposes of supplying energy, and (b) prolonged protein undernutrition. That carbohydrates and fats spare body protein from being catabolized is a fundamental principle in physiological teaching, and it is clearly discussed by Sherman<sup>2</sup> together with a short review of the work upon the subject. Many experiments have been done to reduce the urine nitrogen excretion to an absolute minimum (reviewed by Smith<sup>3</sup>) and they show indirectly the importance of the second

factor in determining minimum protein requirement, since the lowest urinary nitrogen excretion was obtained in those subjects who had been maintained upon a low protein diet for a considerable time.

Rabe and Plaut<sup>4</sup> have conducted an experiment which demonstrates the marked effect of protein and general undernutrition upon protein requirements. Chart I is a copy of their data, and shows that their subject, a normal man of 40 years, weighing 67 kilograms and measuring 168 cm. in height, finally (on the 43rd to 47th days) reached a positive nitrogen balance on 0.21 grams of protein per kilogram of body weight with 0.04 grams of protein per kilogram of body weight retained from the diet daily. The protein catabolism was 0.15 grams per kilogram of body weight and the average daily caloric intake was only 1046. The patient remained mentally and physically active and showed no ill effects despite the fact that during the experiment he had lost in the urine alone, without replacement, the equivalent of 380 grams of protein from his body (approximately 3.0 per cent of the total body protein stores).

It is highly probable that, through protein undernutrition, protein requirement may be reduced considerably below the present accepted minimum figure of 0.6 grams per kilogram of body weight, and without harm.

#### ENDOGENOUS NITROGEN CATABOLISM

It is important to know the source of the endogenous nitrogen catabolism in order to know the seriousness with which the loss of this nitrogen from the body should be considered when not replaced, and if possible to know how to replace it in whole or in part without feeding protein. Of the endogenous nitrogen catabolized by the body, practically all comes originally from body protein or protein precursors. That which is excreted in the stools is nearly all in the form of albuminoid material secreted from the walls of the intestinal tract and may be considered a form of desquamation. The greater the food residue the larger will this fraction be. On a low protein, high carbohydrate diet it averages about 0.7 grams of nitrogen daily. The nitrogen lost in the sweat is in the form of urea and ammonia, and under ordinary conditions is so small as to be negligible. This nitrogen comes from amino-acid catabolism just as does the urine urea and ammonia nitrogen. The endogenous nitrogen lost in the urine has a more complex origin and also may be considered a more important loss. It is only by especially conducted experiments that the nature of this nitrogen can be determined. Just as protein undernutrition lowers the protein requirement of the body, so protein undernutrition lowers the amount of the endogenous nitrogen constituents of the urine. It may be considered that the absolute minimum has not been attained as

yet, but Smith<sup>5</sup> summarizes the work of those investigators who have tried to determine the distribution of the nitrogen in the urine when the minimum nitrogen catabolism was supposedly obtained. The experiment which he presents on a normal adult male of 29 years, weighing 65 kilograms, shows the distribution of nitrogen in the urine when the subject was excreting only 1.58 grams of total nitrogen. This is the lowest twenty-four hour urinary nitrogen excretion in which the nitrogen distribution has been determined. Of the 1.58 grams of total nitrogen, 22.1 per cent (0.35 grams) was urea nitrogen, 14.5 per cent (0.23 grams) was ammonia nitrogen, 35.4 per cent (0.56 grams) was creatinine nitrogen, 7.0 per cent (0.11 grams) was uric acid nitrogen, and 21.0 per cent (0.33 grams) was undetermined nitrogen. There is good reason to suppose that this latter fraction is mainly hippuric acid nitrogen. The food nitrogen for the same day was 0.33 grams, and if this is subtracted from the urea plus ammonia nitrogen (0.58 grams), a true picture of the endogenous nitrogen catabolism is obtained. The urea and ammonia nitrogen then becomes only 20 per cent, and the creatinine nitrogen 44.8 per cent of the total nitrogen. Of these different nitrogen fractions only the urea, ammonia and creatinine nitrogen represents loss from the body musculature. There is some evidence to indicate that this may be replaced by an equivalent amount of creatine added to the diet, but this point awaits further proof.

By appropriate dietary measures it appears quite possible to reduce the endogenous nitrogen catabolism fraction which appears in the urine to 0.02 to 0.03 grams and that of the stools to 0.01 to 0.015 grams of nitrogen per kilogram of body weight per twenty-four hours. Smith was able to obtain this with his subject in twenty-five days.

The experiments of Smith and the authors which he quotes prove that, providing the energy requirements of the body are covered with a diet of carbohydrate and fat, the protein content of the diet may be reduced far below the commonly accepted minimum of 0.6 grams per kilogram of body weight without an increased endogenous protein catabolism. It is only when the body must draw upon its protein stores for energy that the endogenous nitrogen catabolism will increase, and then not even 0.6 grams of protein per kilogram of body weight will suffice to maintain nitrogen equilibrium.

#### EFFECTS OF PROTEIN UNDERNUTRITION

It is not possible to say what injurious effects may be expected from prolonged protein undernutrition when the requirements of the body for food accessory substances and energy are fully supplied. These conditions are seldom fulfilled and there are no published experiments of such

a nature covering many months. Lusk<sup>5</sup> emphasizes the point that with a negative nitrogen balance, it may take as long as three years to deplete the body of one-quarter of its protein stores when these stores are not drawn upon for energy. The reason for this is to be found partly in the fact that with protein undernutrition the protein requirements of the body decrease. This is only one of many adaptive and protective mechanisms which the body is able to exert in time of stress.

The subject of Rabe and Plaut<sup>4</sup>, who had for many years lived on a low protein diet and then submitted himself to a twenty day fast, and twenty-three additional days of low caloric and low protein diet, maintained physical and mental vigor throughout. The subject of Smith<sup>3</sup>, who for twenty-five days remained on an average three gram protein diet, showed no ill effects.

The commonly quoted example of the effects of protein undernutrition is that of "war edema." The literature upon this condition is voluminous, but the general aspects of the subjects are thoroughly reviewed by Lusk<sup>5</sup>. That this is not an example of the effect of protein undernutrition alone is clearly shown; the diet of the many thousands so affected was sadly deficient in both calories and accessory food substances. The inference, that the edema was the result of protein starvation because the plasma protein content was diminished, is not necessarily correct. If it is correct, one must bear in mind that on account of extreme general undernutrition the body protein sources have been so drawn upon for energy that the nitrogen lost is much in excess of that which would have been lost on an even lower protein intake with the total caloric requirements fully satisfied.

As stated at the beginning of this section, it is not possible to estimate what the effects of protein undernutrition will be, or when they will occur, or whether there will be any at all, if the energy and accessory food substance requirements are fully satisfied. There is sufficient evidence to suggest that the effects will be very slight even after many weeks of such a dietary regime. In those nephritics who require such marked dietary protein curtailment that the question of harmful effect arises, the disease will be more apt to prove fatal before any injurious results of protein undernutrition can make an appearance.

When constructing diets in which the protein content is below the "protein requirement," with the intent of bringing the total protein catabolism to a minimum and with the least possible harm to the subject, it is important to satisfy the following prerequisites:

1. The energy expenditure should be covered or exceeded (2500 to 3000 calories) with carbohydrates and fats. The carbohydrate calories should not fall below seventy-five per cent of the total. The free use of jellies, highly sweetened

lemonade and orangeade and cornstarch pudding necessarily form the basis of this caloric supply. Smith<sup>3</sup> gives a recipe for a bread with very little nitrogen content made from cornstarch. This bread is quite palatable when eaten soon after baking. Fat is easily supplied in the form of olive oil salad dressing, salt free butter, and cod liver oil.

2. An abundant supply of vitamins should be maintained in the form of fruits, green vegetables, butter, and cod liver oil.

3. The roughage of the diet should be kept at as low a level as is consistent with a liberal supply of vitamin B, because the excretion of albuminoid material from the walls of the intestinal tract is increased when the intestinal residue is large.

#### SUMMARY

It has been the purpose of this discussion to show that diets of lower protein content than now commonly used in the dietary treatment of chronic nephritis with nitrogen retention are feasible. The amount of protein allowed in the diet should be based upon the amount of non-protein nitrogen which the patient is able to excrete during twenty-four hours. If it is desired that the nitrogen retention be relieved, the nitrogen from the protein in the diet should be less than the total twenty-four hour non-protein nitrogen in the urine. When the twenty-four hour nitrogen excretory capacity of the kidneys equals the amount of endogenous nitrogen catabolism, it will be impossible to relieve nitrogen retention by dietary measures. With properly constructed diets it is approximately accurate to calculate the endogenous nitrogen catabolism at 0.02 to 0.03 grams per kilogram of body weight per twenty-four hours. When any subject partakes of a diet which satisfies the energy requirements in full, but with protein nitrogen content of less than one gram, there is at first a strong negative nitrogen balance and then this gradually diminishes until it approaches a minimum. If such a diet is indefinitely prolonged, it might be possible to deplete the body protein to a dangerous extent. However, there is sufficient evidence to show that such a point would not be reached for many months. This fact makes it practical to administer diets of very low protein content to patients having chronic nephritis with nitrogen retention, for where chronic nephritis has progressed to such an extent as to make necessary diets practically free from protein, other complications of the disease will be more apt to prove fatal sooner than the effects of protein starvation can become apparent. These measures are highly practical in rapidly relieving nitrogen retention, which is the result of excessive protein intake in the presence of a moderate degree of renal insufficiency; after relief of retention a protein intake could be given which would ensure nitrogen balance.



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DIRECTORY FOR WETNURSES

BY FRITZ B. TALBOT, M.D.

HUMAN milk has always been known to be the best food for babies. Its importance has been emphasized in all pediatric textbooks. Infant morbidity and mortality statistics have shown that the baby fed on human milk is less likely to be ill and is more apt to live than those fed on the bottle. The advantage of maternal nursing or feeding with human milk is so obvious that it need not enter into the present discussion. It is interesting to note that the French very recently have encouraged and emphasized the use of human milk and have established stations all over France for the purpose of reducing infant mortality and increasing their population in an attempt to counterbalance their losses during the war.

There is a tendency among those who attain some facility in modifying milk to make it easy for the mother to wean the baby and use cow's milk. Everyone admits that there are a few instances in which it is not wise for the mother to nurse her baby, for example when she has tuberculosis or any chronic wasting disease. But there are unfortunately more cases in which the mother is apparently unable to carry on her duties in her home and also nurse her baby. It is not necessary to comment upon the mother who will not nurse her baby. The blame of weaning the baby cannot always be laid to one door. In many instances, when the doctor recommends cow's milk for young infants, the lack of human milk results in grave malnutrition.

The history of the collection of human milk in this city goes back to about 1910. Before that time when a physician wished to obtain a wetnurse for a baby it was necessary for him to hunt through the various maternity homes for a suitable mother who was willing to go for a compensation into the home of the sick baby and nurse it. Suitable wetnurses were often difficult to find and in many instances they were obtained only when it was too late. If they had been immediately available the life of a baby might have been saved.

The Directory for Wetnurses was established in February, 1910, and its experiences for the first year or two were reported by the writer in the *Journal of the American Medical Association*, 1911, Volume LVI, page 1715. The original purpose of the Wetnurse Directory was to house wetnurses and their children in order to have a constant supply of human milk for sick babies. This proved to be very successful and many babies' lives were saved by the milk

obtained from the Directory. A social factor also entered into this charity because many of the unmarried wetnurses were able to remain with their own children for several months and thus give it a good start in life. They were also able to save money because during their stay at the Directory all their living expenses were covered.

This method of handling the problem was given up because of the overhead expense and the inability of the limited number of women to produce enough milk to supply the needs of both private patients and hospitals. The next move was to have a socially trained public health nurse collect breast milk in bottles from healthy mothers. This proved to be so successful that the original idea of maintaining a directory for wetnurses was given up, the home sold and an office established at 270 Commonwealth Avenue with a supervisor and two nurses who now collect the excess milk produced by mothers in their homes. This milk is brought back to the office, where it is bottled and sterilized ready for delivery. The mothers who produce it are married women who have a negative Wassermann, healthy infants, clean homes, and whose health has been found to be good by physical examination. At the present time there are 35 mothers supplying milk in this way. They are producing nearly 500 ounces a day and during March, 1927, gave a total of 13,000 ounces. There does not seem to be any limit to the amount of milk that can be obtained in this manner and with little notice the supply can always be brought up to the demand.

Obstetricians, the Community Health centers and the maternity hospitals supply the names of healthy mothers to the Directory. The trained nurse then visits and inspects the home and teaches the mother how to express her milk in a sanitary manner. Sterilized bottles are left at each house daily and filled by the mother, placed on the ice and covered with a sterilized rubber cap. These bottles are collected once in twenty-four hours.

The milk has rarely been tampered with and in no instance has cow's milk been substituted for human milk. This is checked up in the following manner:—The chloride content of the human milk is very consistent within normal limits. The chloride content of cow's milk is very high. A sample of the milk of each mother is examined at intervals without the knowledge of the mother. If the chloride content of the

milk is very low it means that the milk has been diluted with water. This has been found in one instance. If the chloride content of the milk is higher than the ordinary variations in human milk it means that cow's milk has been substituted. This has not occurred in a single instance. Bacterial counts are taken at intervals. Before bottling, the milk is strained, pooled, then boiled for one minute, allowed to cool, then re-strained and bottled. Model sterilizers, bottle washers and refrigeration plants are being installed to take care of the increased supply of milk. The milk, when ready for delivery, is packed in ice in cases.

At the present time about two-thirds of the milk is supplied to private patients and one-third to hospitals. The well-to-do pay twenty-five cents an ounce for this milk. In needy cases a reduction is made. The hospital pays twelve cents an ounce, which, with the present bulk of production, is very close to or slightly below cost. If the demand of hospitals becomes greater, it may be possible to supply milk to them at an even greater reduction in price.

*The use of human milk:*—Every hospital caring for infants in Boston has babies that are critically ill whose chances of life are greatly increased when they receive human milk. Among the conditions so treated during the past year at the Massachusetts General Hospital are broncho-pneumonia, diarrhoea, dehydration, acidosis, prematurity and congenital stenosis of the pylorus. All infants with surgical conditions have a much better chance on human milk. This was appreciated by the surgeons at the hospital a hundred years ago for they gave those patients who had been nursed by their mothers a better prognosis than those who had not.

In private practice it is not uncommon to give a newborn infant milk from the Directory for the first few days until the mother's milk comes in. In other instances human milk has been given when the formula could not be made to agree, or after a baby was upset by a proprietary food. In some instances babies, whose mothers could not nurse them for one reason or another, have been fed on this milk for nearly a year because the parents wanted to give them the best possible start in life. It's use in the case of the premature infant has been invaluable and undoubtedly has been in large part responsible for the excellent results seen in these cases. One infant at present receiving this milk gained from a birthweight of 4 lbs. 8 ounces to 6 lbs. 8 oz. in eight weeks time. This case can well be contrasted with another premature infant fed on modified milk when human milk could not be obtained, and gained only eight ounces in four months and required two trained nurses throughout the entire time.

If the number of children whose lives have been saved by the use of human milk were

known, there is little question that similar Directories would be established in every city of the United States.

### SUMMER CLINICS, CHICAGO MEDICAL SOCIETY, 1927

ANNOUNCEMENTS and schedules will soon be ready for the 1927 Summer Clinics of the Chicago Medical Society, supported by many of the largest hospitals in the city, among them being the Post Graduate Hospital, Chicago Memorial Hospital, University of Illinois College of Medicine, Cook County Hospital, Michael Reese Hospital, Mercy Hospital, Presbyterian Hospital, Jackson Park Hospital, St. Luke's Hospital, Ravenswood Hospital, Mount Sinai Hospital, Francis Willard Hospital, West Suburban Hospital, Evangelical Hospital, North Chicago Hospital, Chicago Lying-in Hospital, St. Joseph Hospital, Alexian Brothers Hospital, Laboratory of Surgical Technique, Washington Park Hospital, Jackson Park Hospital, Chicago Municipal Tuberculosis Sanitarium, John B. Murphy Hospital. Several of our large laboratories have also agreed to coöperate with us in this great work.

In 1926 registrations were limited to physicians living in Illinois, but increased facilities make it possible to accommodate many more than last year. Registrations therefore will be open to physicians from other states. Registration fee will be \$10 for each two weeks course.

The first two weeks course will begin on Monday, June 13th, 1927, at 9 a. m., ending Friday, June 24th.

The second two weeks course will begin on Monday, June 27th, at 9 a. m., ending Friday, July 8th.

### THE CENTENNIAL OF THE BIRTH OF SIR JOSEPH LISTER

On April 5th, 1927, there were celebrations in England and other places of the centennial of the birth of Sir Joseph Lister, the father of antiseptic surgery.

Lord Lister trained in medicine in England practiced under the noted surgeons in Scotland. In connection with hospital work he was saddened by the large loss of life from wound infections which should normally have yielded to treatment. So he put into practice the cleansing of wounds by an antiseptic to avoid further spread of such infections. This led to great saving of lives.

So we owe a debt of gratitude to Lister who paved the way for our present universal practice of aseptic surgery through whose principles nothing comes in contact with the wound unless it is first made absolutely sterile.

**Case Records**  
of the  
**Massachusetts General Hospital**

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN  
WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.  
F. M. PAINTER, A.B., ASSISTANT EDITOR

**CASE 13161**

**A CASE OF COMA—DURATION ONE  
WEEK WHEN FIRST SEEN**

**MEDICAL DEPARTMENT**

A married American woman forty-nine years old entered the hospital January 28 in coma. The history was given by her husband.

Two years and a half before admission when apparently in good health she one day became very short of breath after walking a mile and climbing a hill, and had an "asthmatic attack." (According to another account her symptoms began at the birth of her ninth or tenth child.) A physician gave morphia with considerable relief. After this she was comparatively free from symptoms except for occasional violent pounding of the heart until a year before admission. Then she began to have occasional headaches and marked throbbing in the head at night. One morning she found she could not turn over in bed, and later could not use her right arm and dragged her right foot. After a month she completely recovered, was quite well and able to walk a mile without discomfort, though for a year she had urinated twice at night. Eight months before admission she suddenly fainted, and on regaining consciousness had another bad "asthmatic attack." She was given morphia with some relief and put to bed for a week. On the first day she had a generalized convulsion lasting five minutes with unconsciousness and twitching. This was not followed by any muscular disturbance. After this she was not allowed to do housework. When she did a little against rules she became very short of breath and tired very easily. For a month she had had edema of the ankles off and on. A week before admission she suddenly lost consciousness and had another five minute convulsion. A physician gave her morphia. She seemed semicomatose. Three hours later she had another convulsion. "Her left blood pressure was 262." The next day she was very drowsy and semistuporous, although she could recognize people. The day before entering the hospital she had marked orthopnea and could sleep only sitting up. At four o'clock the morning of admission she became irrational.

Her mother died of possible Bright's disease. The patient's past history is negative except for

diphtheria in childhood and two miscarriages out of thirteen pregnancies. Eight children were living and well. She had never had a doctor except for childbirth.

Clinical examination showed a pale, emaciated, dehydrated old woman, semicomatose, not irrational, breathing stertorously. Teeth poor. Pyorrhea. Tongue dry and furred. Apex impulse of the heart felt in the sixth interspace. Percussion measurements: left border 11 centimeters, midclavicular line 8 centimeters, right border 2 centimeters, supracardiac dullness 4.5 centimeters. Loud systolic and diastolic murmurs at the apex, a soft systolic at the base, perhaps the same murmur transmitted. Artery walls thickened and tortuous. Blood pressure 210/105 to 152/87. Lungs negative except for dullness at the left base. Abdomen not remarkable. Pelvic examination negative except for slight erosion of the cervix. Extremities, pupils and reflexes normal. Fundi: arteriosclerosis, discs slightly blurred, no evidence of choked disc.

Urine 28 to 63 ounces, specific gravity 1.008 to 1.014, alkaline at 2 of 7 examinations, the slightest possible trace to a trace of albumin at 6 of 8 examinations, leucocytes at all of 6 sediment examinations, loaded with leucocytes at 5; no red cells. Blood: 16,800 to 6,400 leucocytes, polynuclears 65 per cent., hemoglobin 55 to 30 per cent., reds 2,348,000 to 1,888,000, smear normal. Non-protein nitrogen 125 to 159. Blood calcium 7.8 to 6.8, chlorides 621 to 615, phosphate 11 to 5.6, uric acid 6.7. Wassermann negative.

January 29 the patient had three convulsions. She was given 15 cubic centimeters of 5 per cent. calcium chloride in 500 cubic centimeters of 5 per cent. glucose intravenously at 10 a.m. and 20 cubic centimeters of calcium chloride in 1000 cubic centimeters of glucose at half past six p.m. A stomach wash was done at 9 a.m. She had no more convulsions that day. Calcium chloride in glucose was continued daily. January 31 she was a little clearer mentally. February 3 she had two more convulsions, one lasting nearly an hour. February 5 another lavage was done. She became much more rational and ate well. February 21 she was able to be up and about the ward. March 2 she was sinking back into coma. An abscess on the neck was opened and drained. March 3 she died.

**DISCUSSION**

BY RICHARD C. CABOT, M.D.

**NOTES ON THE HISTORY**

Presumably this measurement of 262 was a systolic blood pressure.

On the basis of the history without physical examination the symptoms suggest the heart and brain. We know that she had high blood pres-

sure on one occasion. We should have suspected it from the pounding headache and dyspnea on previous occasions. She has also had attacks called "fainting attacks" in some of which she is known to have had convulsions, and one of which was followed by hemiplegia which, however, cleared up wholly. In any case suggesting cardiovascular trouble in which fainting attacks with or without convulsions supervene one always wonders whether they may not have been attacks of heart block with Stokes-Adams syndrome.

The other question is whether the high blood pressure itself, with poor heart action and very possibly some cerebral arteriosclerosis, will account for the cerebral symptoms without making any direct connection with heart block. It has to be recognized that the only known record of blood pressure so far was close to a time when there had been cerebral disturbance of a type that is capable of being associated with high blood pressure even when the heart is normal.

So with those possibilities in mind, chiefly of cardiac disease with associated hypertension, and of cerebral arteriosclerosis, we come to the physical examination.

#### NOTES ON THE PHYSICAL EXAMINATION

They say she is an old woman, yet according to the record she is only forty-nine. Presumably therefore she is older physically than her age showed, or else, as so frequently happens in the "nines," it is an artificial age. There is an enormously greater number of people who are twenty-nine than of those who are thirty, a greater number who are thirty-nine than forty or thirty-eight. There is a great heaping up of the "nines." One of Barrie's plays deals with a woman who had "a long twenty-nine" which lasted for years and years. This may be of that type.

There is some suggestion of a limited range of specific gravity in the urine.

This is a very surprising statement about the blood. We very rarely see reds 1,800,000 with a normal smear, but that is the statement.

The non-protein nitrogen, blood calcium, chlorides, phosphate and uric acid all show very high values.

In the two weeks between the 5th and the 21st of February a great deal went on. I suppose they may have thought she was getting well.

The abscess very likely was the precipitating cause of the coma.

#### DIFFERENTIAL DIAGNOSIS

Physical examination gives us evidence of an enlarged heart, presumably connected with high blood pressure. At the first examination they mentioned a diastolic murmur. They did not mention it again. My experience is that when diastolic murmurs are heard only at entrance to a hospital they usually turn out to be unimpor-

tant. Of course there is considerable risk in that inference, but I think I should say that on the whole there is no reason to suppose there is any valve disease here. We have an enlarged heart then, with high blood pressure.

The other two important facts of the case are (1) an intense anemia presumably connected with (2) the nephritis which is the third feature in the case, and which I think we must infer from the persistently low specific gravity, the high retention of products normally excreted, and the pretty steady albuminuria. On the whole it seems to me better to call her convulsions uremic and her anemia secondary to chronic nephritis.

Chronic nephritis, then, with a hypertrophied and dilated heart, with arteriosclerosis presumably, and with no good reason to suppose that gross organic lesions will be found in the brain. It is the sort of case where the brain may show arteriosclerosis and nothing else, where it might show foci of softening, or might show not much of anything.

Her asthmatic attacks, dyspnea and edema I should connect with her cardiovascular trouble, perhaps also with the renal trouble. There certainly is a renal dyspnea as well as a cardiac dyspnea. I suppose the treatment she received here was on the basis of an acidosis, though it does not seem to have had any brilliant result.

#### CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Hypertension.  
Chronic nephritis.  
Uremia.  
Submental abscess.  
Incision and drainage of submental abscess.

#### DR. CABOT'S DIAGNOSIS

Hypertension.  
Chronic nephritis.  
Hypertrophy and dilatation of the heart.  
Arteriosclerosis.  
Secondary anemia.  
Uremia.

#### ANATOMIC DIAGNOSES

##### 1. *Primary fatal lesion*

Arteriosclerotic nephritis.

##### 2. *Secondary or terminal lesions*

Hypertrophy and dilatation of the heart.  
Chronic passive congestion, slight.  
Slight arteriosclerosis.

##### 3. *Historical landmark*

Operation wound.

DR. RICHARDSON: We were not permitted to examine the head. The anatomical conditions in this case were clear and to the point. The pa-



tient was a fairly nourished white woman said to be forty-nine.

DR. CABOT: Did she look much older?

DR. RICHARDSON: It did not strike me one way or the other. I did not make a note of it, and I should have if it did.

There was a slight brownish discoloration of the skin, but nothing that we could call icterus. The peritoneal cavity and the appendix were negative. The gastro-intestinal tract and the mesenteric and retroperitoneal glands were negative. The anterior margin of the liver was five and a half centimeters below the costal border. The diaphragm on the right was at the fourth interspace, on the left at the fifth interspace.

There was practically no fluid in the pleural cavities, and there were no pleural adhesions. The trachea and bronchi contained much pale reddish frothy fluid. The bronchial glands were negative. The lung tissue showed no areas of consolidation and was otherwise negative except for slight congestion.

The pericardium was negative, no fluid. The heart weighed 510 grams, with a thick myocardium, four millimeters on the right, fifteen on the left, and thick columnae carneae. The cavities on the left were of full size and on the right showed slight dilatation. The valve circumferences were: mitral 8.5 cm., aortic 6.5 cm., tricuspid 12 cm., pulmonary 7.5 cm. The valves were frankly negative. The coronaries were free. The aorta showed scattered along it a small amount of sclerosis, generally fibrous in character but in a few places calcareous. The branches were negative. So that the amount of sclerosis present was comparatively slight.

The liver, gall-bladder, pancreas, spleen and adrenals were negative.

The kidneys weighed 89 grams,—marked atrophy. The capsules were slightly adherent, the surfaces pale gray-red and granular, and the tissue granular, tough, with very indistinct markings. The cortex could not be made out definitely. That is, of course, a typical picture of chronic nephritis. Such kidneys would rest comfortably in the cavity of the left ventricle. Microscopically it was arteriosclerotic nephritis.

The microscopic examination of the myocardium was negative. The pelves, ureters and bladder were negative, as were the uterus and adnexa. The amount of chronic passive congestion was very slight.

There was a submental abscess, but no infection of the blood stream. The culture was frankly negative.

DR. CABOT: One speculates a little on the genesis and order of events in a case like this. Twenty years ago we should have said: of course that is perfectly simple; the woman got chronic nephritis; as a result of that the heart hypertrophied and dilated; she died of uremia. Now I suppose we should say that is a possible explanation. It is more likely however that the hypertension

came first and the kidney trouble developed later, or the two developed simultaneously as a result of some unknown cause back of them both. We certainly see more and more cases of hypertension without nephritis in its earlier stages, and of hypertension with nephritis later. It is a natural though not necessary conclusion that the hypertension leads to the nephritis rather than vice versa as we used to say.

This seems to be the sort of case in which if Dr. Richardson had opened the head, there being so little arteriosclerosis elsewhere, he probably would not have found anything there. I think it is important to remember a case like this, when the question of hemorrhage or other gross lesion in the brain is raised because of hemiplegia, because of coma, because of convulsions, that there are cases with these symptoms in which examination after death does not show anything, and it is as a result of the hypertension or of the uremia that we have these apparently focal manifestations. It is in this sort of case that the convenient hypothesis of vascular spasm in the brain is advanced, the difficulty about it being that we can never either prove it or disprove it. By the time we get there if there has been any spasm it will have relaxed. So it is not the kind of hypothesis that leads to any further knowledge.

It occurs to me to say here, with regard to these exercises which have gone on for so many years, that questions have been asked whether we know about these diagnoses before we come here to discuss them. There are a good many such exercises conducted in other parts of the country, especially at Johns Hopkins, where they make a point of knowing and carefully studying the necropsy beforehand in order that they may give a better presentation of the case, knowing all about it. But it is not the way we do it here. We make a practice of standing or falling on pure reasoning. Of course we also have here cases which have got well, in which the clinician argues on the basis of known facts; also cases in which he knows the post mortem and argues with that definitely in mind. But it ought to be understood that when we do not say that, those of us who present cases here are doing it without any such knowledge. I may say that I used to look at the necropsy and then tried to argue just as I would have if I had not known the necropsy. But I inevitably pushed my reasoning towards what I knew was there. I could not be honest, and so I gave it up.

#### CASE 13162

#### A CASE OF PROGRESSIVE SECONDARY ANEMIA

#### MEDICAL DEPARTMENT

On January 11 an unmarried Swedish cook forty-six years old entered a private hospital

complaining of weakness and respiratory pain on the left. Two months and one-half before entrance she had noticed increased thirst and frequency of urination. She became irritable and lost strength. She continued thus for three weeks, when she developed an acute respiratory infection with phlebitis in the left lower leg. At the same time she had what seemed to be pleurisy in the left chest. She was in bed for three weeks during December with fever. The leg quieted down, but she still had pain in the left chest. She did not complain of thirst or polyuria on entrance. She did however, complain of buzzing in the left ear which bothered her a good deal and which had been present on and off for two months.

The family history was not important except for the fact that one sister had epilepsy. There was no diabetes, cancer or hemophilia.

Her previous health had always been good. No infectious diseases were recalled. She had had no symptoms indicating disturbance in the cardio-respiratory, genito-urinary or gastro-intestinal systems. She had had varicose veins in her left leg for ten years. At the age of seventeen she weighed 120; at the age of nineteen she started to gain weight and had gained steadily. Her weight on entrance was 213½ pounds without her clothes. She had always been a cook and fond of bread and butter, but denied being a "heavy eater."

Physical examination showed marked obesity and pallor. There was puffiness of the upper and lower eyelids. The pupils were equal and reacted to light and distance. The teeth showed a great deal of dental work and many gold caps. No glands were palpable. The sinuses and throat appeared normal. The heart was not enlarged to percussion. The sounds were normal. The pulse was rapid, varying from 90 to 100, and at times there was an irregularity thought to be due to extracardiac systoles. There was no definite abnormality in the lungs. The blood pressure was 160/100. During her stay in the hospital a very large spleen and an enlarged liver were found. There was no evidence of ascites, and no other masses were found. The knee-jerks were sluggish.

On entrance her urine contained 5 per cent. of sugar, no diacetic acid, the slightest possible trace of albumin and a few pus cells in the sediment. The specific gravity ranged from 1.030 to 1.010. She was considered at first to be a mild diabetic who would yield to ordinary treatment, but it was found after two weeks that although she became sugar-free on 45 units of insulin daily, the fasting blood sugar remained at a level between 250 and 300 milligrams per 100 cubic centimeters. The temperature rose daily to 99.5° or 100° and the pulse varied between 90 and 110. The non-protein nitrogen was 30 milligrams per 100 cubic centimeters, the bilirubin 0.2 and 0.3 milligrams per 100 cubic centi-

eters. During the month of February she continued running a slight degree of fever, was sugar-free on 45 units of insulin a day, still maintaining a high blood sugar. She complained a good deal of buzzing in her head, general weakness and pain in the left chest on respiration. A nose and throat consultant reported as follows:

"History suggests a recurrence of an acute catarrhal condition of the left middle ear, though there is no clinical evidence of it now. The deafness seems more than would be expected with this condition, but the patient states that her hearing has been even worse and not relieved by catheterization. Throat negative except for some hypertrophy of the tonsils. Nose shows an irregular deviated septum, but no obstruction or evidence of sinus infection."

A surgical consultant reported as follows:

"The left lower rib three inches from the sternum is slightly swollen and definitely tender. The tenderness is most marked in the most prominent part, but pressure over the attachment to the sternum is painful. This may be tuberculous, but is more probably periosteal. The history of soreness following a cold of one month ago is suggestive of a metastatic infection. I do not believe it is of the nature of a new growth. Probably an osteoperiosteal infection following an acute general infection. No definite treatment indicated at present."

A gastro-intestinal X-ray series showed nothing abnormal except displacement of the intestines by the spleen. X-ray of the chest showed evidence of an inflammatory process about the hilus of the right lung, but otherwise nothing. The blood Wassermann was negative. The leucocyte count was 8,000 with 60 per cent. neutrophils, 34 per cent. lymphocytes, 3 per cent. large mononuclears, 2 per cent. basophils and 2 per cent. eosinophils. The reds showed a great deal of basophilia with well marked stippling; numerous blasts and a good deal of deformity, with numerous microcytes. There was well marked achromia. The polychromatophilia was striking. The blood platelets were practically absent (artefact?). There were all kinds of blasts—normoblasts, megaloblasts. It appeared to be a very severe secondary anemia and seemed hemolytic. The red count was 2,200,000 and 2,400,000, with a hemoglobin by Tallqvist of 50 to 55 per cent. The blood culture was negative in four days.

Following the two X-ray pictures of the chest the pain in the chest decreased and the small nodule felt on the rib disappeared.

She continued to go downhill, with increasing weakness, dyspnea and edema. There were no purpuric spots found, and she was not jaundiced. She remained sugar-free on 45 units of insulin a day, maintaining a persistently high blood sugar. She was drowsy and comatose up to March 11, the day of her death, which was

preceded by a profuse hemorrhage from the nose.

#### DISCUSSION

BY WILLIAM B. BREED, M.D.

#### NOTES ON THE HISTORY

This patient was referred to the hospital as a diabetic. She was a very fat woman, and was assumed to have mild diabetes complicated with acute respiratory infection of some sort as well as with phlebitis. That was the first impression, and that is what we should get from the history of the present illness.

The history of varicose veins would fit in with the flare-up of phlebitis during an infection, which would temporarily make her mild diabetes more severe.

Six months before her entrance she had weighed 240 pounds, and on entrance her weight was  $213\frac{1}{2}$  without her clothes. She was a cook and admittedly fond of bread and butter. This is a very common history in obese people, especially those who develop diabetes. Of course we might have looked for some endocrine gland disturbance to account for this obesity, but with the development of the diabetes it did not seem necessary to go as far as that, because the history at least rather clinched the diagnosis of obesity on the basis of overeating.

#### NOTES ON THE PHYSICAL EXAMINATION

She was a short woman, and  $213\frac{1}{2}$  pounds was extreme obesity.

So far as the history and physical examination go, without the extreme anemia and the presence of the enlarged spleen and liver, she was a definitely mild diabetic who had developed some infection and who should have yielded to ordinary treatment without the use of insulin, or perhaps with the use of insulin until the infection had quieted down.

But while on paper she was a mild diabetic, during her stay in the hospital she did not appear like a true diabetic. She did not have the symptoms of diabetes, and the progress of the case while she was there made us turn from diabetes and focus on the blood condition.

We were unable to infer that she had any kidney damage.

She became sugar free easily with the use of forty-five units of insulin, which is rather a high dosage. But we were never able to reduce the blood sugar below 250. It varied from 250 to 350 milligrams in the blood in spite of her being sugar free as far as the urine was concerned. The normal threshold is about 200 milligrams in the blood before the sugar shows in the urine. Before the days of insulin there were a few very severe cases that remained sugar free with a blood sugar of 250 milligrams, but rarely higher than that, and they were unusual cases.

The non-protein nitrogen is normal.

The bilirubin was taken after the blood had been worked up more thoroughly, and is a normal figure, the normal being from zero to six-tenths of a milligram. Toward the end of her stay it became apparent to us that her head symptoms were due more to a severe degree of anemia than to a local ear condition. The blood picture was a peculiar one. The differential count is essentially normal. The examination was repeated and the platelets were absent.

The hemoglobin makes the color index slightly over fifty per cent., but I personally do not place much faith in the Tallqvist report when it is as low as fifty, and I think that any conclusion as to the color index from these figures is inaccurate.

"The small nodule felt on the rib disappeared." That was a very interesting observation which was accentuated in this history after we had found out what the patient died of.

As she stayed in the hospital we turned away from the diabetes, which seemed to be of secondary importance. The blood sugar remained persistently too high with that dose of insulin, which did, however, keep the urine sugar free. We rather ignored the diabetes, keeping her, however, on a diet calculated to be sufficient for maintenance.

#### DIFFERENTIAL DIAGNOSIS

We tried to make a diagnosis before death, but were unable to do so. We considered Banti's disease, but we thought that in the first place the blood picture did not fit the syndrome and the progress was far too rapid for that disease. If the history is correct she went downhill very rapidly in the course of three months, and died. Before we did the bilirubin in the blood, believing that the blood showed an anemia of the hemolytic type, we considered endocarditis, but threw that out on the basis of the rapidity of progress, the negative blood culture, and the absence of heart murmurs under observation. We considered malignant disease of some unusual sort, but were unable to determine a primary focus.

On the basis that this was the blood of a secondary aplastic anemia, in view of the normal bilirubin we came to the conclusion that she must have some tremendous over-activity of the bone marrow due either to a tumor of the bone marrow or to some other cause.

In summing up the whole case the interest it seems to me lies in the fact that the anemia, which was at first thought to be hemolytic, turned out to be a severe non-hemolytic secondary anemia. The diabetes, if it was diabetes, was very unimportant. We really made no diagnosis as to the cause of death except that we did believe there was over-activity of the bone marrow.

After Dr. Warren has finished I should like

to be sure to call attention to the observation of the disappearance of the nodule on the chest.

Hemorrhage from the nose was caused by a blow while she was sitting in bed. It was also true that both her arms had ecchymotic spots in the region where she had had her injections of insulin.

A PHYSICIAN: What was the cause of her death?

DR. BREED: I think the hemorrhage precipitated her death, but I think she died of anemia, whatever the cause of that anemia was. There was some over-activity of the bone marrow which caused this very severe non-hemolytic anemia. She did not have purpura I believe.

DR. CABOT: I should not think so. We have no good observations on the platelets.

DR. BREED: They were practically absent. In another smear that I looked at they were very low.

A PHYSICIAN: Was that first count made on admission, or all the way along?

DR. BREED: That was, I should say, when she had been there two weeks. There was no demonstrable change in the lymphatic cells or the myelogenous blood system. It was a red blood cell disturbance as far as we could tell.

DR. CABOT: I cannot make a diagnosis.

DR. SHIELDS WARREN: This woman was very obese. I estimated her weight at 275 pounds. The head was of normal contour, with abundant brown hair. The pupils were equal and regular, six millimeters in diameter. The chest was symmetrical. The breasts were very fat and contained a few small masses which on section were found to be thin-walled cysts. Rigor mortis was marked, and there was rather marked pallor of the entire body. The external genitalia were negative.

When we opened the abdominal cavity we found it to contain a small amount of thin blood-tinged fluid, about 150 cubic centimeters. The surfaces were smooth and shiny. The appendix was negative. The mesenteric lymph nodes were very hard to find but were apparently not enlarged.

The pleural cavities each contained about a liter of thin blood-tinged fluid. There were numerous thin fibrous adhesions between the parietal and visceral surfaces of the pleura, more marked at the bases. There were several smooth nodular swellings on the inner surface of the chest wall, apparently firmly adherent to the periosteum of the ribs. They measured 3 by 1.5 centimeters. On section they were pale pink, rather uniform in appearance, and fairly soft. The under side of the sternum as it was turned back was plastered over with a fairly firm dark red material, apparently a hemorrhagic mass, perfectly smooth, and beneath the mesothelial covering of the pleural cavities. This mass was

about one millimeter thick at the edges and one centimeter deep in the central portion.

The pericardial cavity was negative. The heart weighed 270 grams and was negative except that the myocardium was relatively flabby and of an opaque yellowish brown appearance, suggesting a certain amount of fatty change. The valves and coronary arteries were negative.

The lungs were pale red, soft, crepitant throughout, with the usual anthracotic markings on the surfaces. On section they were pale and fairly dry except that toward the bases a little thin red fluid could be expressed. The trachea and bronchi were negative.

The spleen weighed 1500 grams. There were a number of fibrous adhesions between the peritoneal covering of the spleen and the peritoneal wall. On section it was soft and purple red in color. All normal markings were obscured, and when lifted up the pulp dripped from the cut surface.

The gastro-intestinal tract was essentially negative. The pancreas weighed 140 grams, but that was no index of the amount of pancreatic tissue present, because there was extensive fatty ingrowth. The adjacent retroperitoneal lymph nodes were very much enlarged, 3.5 centimeters in diameter, and on section were soft and meaty. The liver weighed 5400 grams, tremendously large. The capsule was smooth and the liver rather pale and an opaque yellowish color. On first examination I felt it was probably the type of fatty liver found in obese individuals. On section it cut with much resistance. The markings were distinct. The periportal areas stood out particularly. The knife was decidedly greasy. The bile-ducts were patent. The gall-bladder was thin-walled and contained two small brownish stones a half centimeter in circumference, the typical cholesterol gall-stones.

The left kidney weighed 175 grams. It was fairly adherent to its bed. The capsule stripped easily, leaving a smooth surface. On section there was seen to be a red fleshy mass between the pelvis and the kidney tissue varying from 0.3 to 1.5 centimeters in diameter. At no point had it ulcerated into the pelvis, but rather insinuated itself between the parenchyma and the epithelium of the pelvis. The drawing shows the dark red mass presenting under the pelvic epithelium and extending out without distorting or invading the kidney. We may also notice the great thickening of the ureter. This red mass could be seen extending beneath the pelvis, and its edge was decidedly hemorrhagic in character. The cortex of the kidney was 1.8 centimeters thick. The glomeruli were visible, the pyramids not particularly well marked.

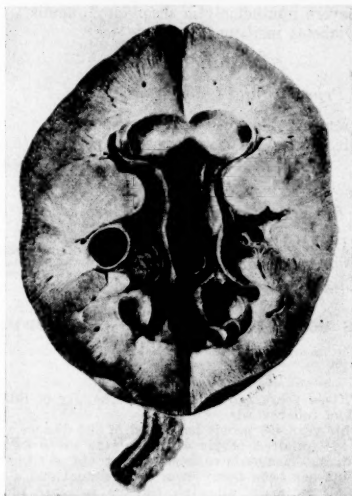
This same description holds true for the right kidney, with the exception of its weight, which was 225 grams, and the fact that the tumor mass was slightly more extensive.

Both ureters were encased in a reddish firm mass continuous with that described in the kidneys, and extending the entire length of the ureters into a mass which became merged into the pelvic muscles. There was no obstruction and no ulceration of the tumor growth into the lumina. The adrenals were essentially negative.

The bladder contained about 200 cubic centimeters of rather cloudy slightly foul urine.

The cervix showed a few small cysts filled with translucent fluid. The uterus was negative both as regards musculature and mucosa. The ducts and ovaries were negative.

The entire pelvic musculature, however, in-



Left kidney. A dark red mass presents under the pelvic epithelium, extending out without distorting or invading the kidney. The ureter is greatly thickened.

From a sopia drawing by Miss Etta R. Plotti.  
DR. SHIELDS WARREN.

cluding the psoas, iliacus, and the levator ani was merged into a hard mass which was entirely retroperitoneal and was continuous with the growth described about the ureters. There was no continuity with or invasion of the rectum or the other pelvic organs.

The retroperitoneal lymph nodes from the pelvic floor to the diaphragm were enlarged, and in most cases the enlarged lymphatic vessels could be seen between them. Some were hard and white, others very firm and dark red, while others were pale, soft, pink and friable. The mediastinal lymph nodes showed a similar varied change and apparently were involved in the same process as those below the diaphragm.

The aorta showed a moderate number of

atheromatous plaques in the abdominal portion. One of these was calcified.

The bone marrow of the ribs was a pale pink and apparently of the usual character. That of the bodies of the vertebrae (three were examined) was of a somewhat darker red than that of the ribs, but showed no apparent variation from the normal.

When I finished the necropsy I had no idea what I was dealing with, and even a frozen section did not give much light. But on histological examination of the kidney pelvis we found a very interesting thing—essentially hemorrhagic tissue consisting of a few megakaryocytes, eosinophils, myelocytes, with many red cells of all sizes and stages of development ranging from normoblasts to megaloblasts. In addition there were large numbers of undifferentiated cells which as nearly as we could tell were the primitive blood-forming cells, Maximow's hemocytoblast. The spleen and liver were infiltrated by a similar type of cell. In these two organs there were fewer of the more adult forms and more of the relatively undifferentiated cells. This cell is a large cell with a vacuolated nucleus and a fair amount of pale cytoplasm. With methylene blue it tends to take a basic stain.

The lymph nodes showed the same general structure. The pelvic musculature was largely destroyed and replaced by a similar tissue.

The most disappointing thing was that the bone marrow was essentially that of a secondary anemia. In trying to discover some clue to the region where this tumor might have arisen I found an article by Jordan\* showing that in the lower animals, particularly in the frog and the tadpole, the pelvis of the kidney is the prime blood forming organ; and it seemed possible that the growth started in the region of the pelvis of the kidney. I neglected to say that occasionally we found a few spicules of bone and fat cells, making the similarity to bone marrow even more striking. Apparently there is an opportunity for bone marrow formation in the kidney in man. A number of cases have been reported in splenic anemia and in some of the cases of myeloid leukemia and acute leukemia found in infancy. So far as I know there is no tumor with which this can be compared. I have sent slides to a number of pathologists, who feel as I do that it is one of the pathological curiosities which we occasionally find, and apparently represents a tumor of the primitive blood cell. Dr. Mallory said we might call it a hemocytoblastoma.

A PHYSICIAN: Was a Wassermann taken?

DR. BREED: The Wassermann was negative.

A PHYSICIAN: You did not find anything in the bone?

DR. WARREN: No. It was simply the usual type of marrow, with a rather marked second-

\*Jordan, A. G., and Spield, C. C., American Journal of Anatomy, 1923, Volume 32, page 155.



dary anemia. Dr. Peabody made the suggestion that the tumor might be primarily of the red blood forming cell, but he felt that he could not tell the difference between that and the hemocytoblast.

A PHYSICIAN: Was that nodule on the rib found at necropsy?

DR. WARREN: Yes. We looked there and there was a little fibrous thickening. There were nodules on the under surfaces of the rib that were apparently similar to the one that had been felt.

Isn't it rather unusual for a definite tumor nodule to yield to such a brief X-ray exposure as one would get in two X-ray photographs? This was on the front a little below the breast.

DR. CAMP: I do not see how she could possibly get enough X-ray from a picture of that sort.

DR. BREED: On looking back it occurred to us that that could be the only explanation of the disappearance of that nodule and the patient's symptoms, and that this being such a very young cell might be more susceptible to X-ray than the blood tumor that we are familiar with.

DR. RICHARDSON: Did these nodules that you found have the same tumor tissue in them?

DR. WARREN: They had the same histological character and they were not continuous with the marrow of the ribs. This one that disappeared was on the outside and simply showed as a fibrous thickening of the periosteum on the outside of the anterior surface of the sixth rib.

DR. CABOT: Isn't the gross situation of this tumor down in the pelvic muscles very queer?

DR. RICHARDSON: Yes. I never saw that and never heard of it.

DR. CABOT: As I get it the pelvis contained the largest single tumor outside the liver and spleen?

DR. WARREN: All parts of it were connected. That is, it seemed to have spread by extension rather than by metastasis. We could trace its passage down the ureters into the pelvic mass. The lymph channels between the nodes were enlarged and continuous up to the mediastinum and with the mass spread out over the under surface of the ribs.

DR. RICHARDSON: You think, then, it had followed the lymphatics much as lymphoma would do? The picture of the kidney and liver tissue is something like lymphoma or blood tumor.

DR. WARREN: Yes, it is very similar.

DR. CABOT: Was it red?

DR. WARREN: Yes, in places very hemorrhagic. The ureters when cut across were quite a dark red. It could be seen from the pelvis of the kidney as dark red, and under the sternum it was distinctly hemorrhagic. In some of the lymph nodes it was pale and friable. Where there was a large proportion of the lymph cells

it was pale. Where there were red cells it was hemorrhagic in character.

DR. BREED: I understood you to say it was less likely to be the red blood forming cells than the lymphatic or the myelogenous cells.

DR. WARREN: It is hard to say whether it is a primitive red blood forming cell or whether it is the ancestor of all the blood cells. In almost any given field one could find representatives of cells that could pass for either the myelogenous or the lymphatic series.

#### CLINICAL DIAGNOSIS

Unknown cause of over-activity of the bone marrow.

Severe non-hemolytic secondary anemia.  
Diabetes mellitus?

#### ANATOMICAL DIAGNOSIS

##### 1. Primary fatal lesion

Hemocytoblastoma of kidneys.  
Metastases to ureters, pelvis, retroperitoneal and mediastinal lymph nodes, liver and spleen.

##### 2. Secondary or terminal lesions

(Diabetes.)  
Bilateral hydrothorax.  
Cholelithiasis.

#### HAS MODERN TUBERCULOSIS CRUSADE PAID THE COMMUNITY?

BY ALBERT H. GARVIN, M.D.

Fifteen years ago 850 people in the city of Buffalo died of tuberculosis.

This year 450 people have died of the disease.

Four hundred people are this year alive who 15 years ago would have been among the dead. The decline has been fairly even and gradual and a reasonable estimate, allowing for the increase in the size of the city, is that there are at least 4,000 people alive today who under older methods of care would be counted lost.

The value of life is increasing. An old estimate placed a life at the value of \$5,000. A life insurance company recently placed the actual cost of bringing up a child to adolescent self-supporting age at more than \$17,000.

The saving at present costs amounts to \$68,000,000. This gross saving has resulted from a capital and maintenance expense of \$12,000,000 during the period mentioned, leaving a net saving of \$54,000,000. As realizable assets there are still left the complete plant at Perrysburg and the tuberculosis buildings at the City Hospital, which should be deducted from the "Expense to save." When these places may no longer be needed for tuberculosis they are available for other great untouched problems: The convalescent care of the acute surgical and medical case; the heart problem; the post-poliomyelitis problem, and others.—*Bulletin of the Buffalo Department of Health.*

#### WEEKLY HEALTH INDEX SUMMARY

Telegraphic returns from 68 cities with a total population of 30,000,000 for the week ending March 26, indicate a mortality rate of 13.2 as against 19.1 for the corresponding week of last year.

## THE BOSTON Medical and Surgical Journal

Established in 1828

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

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SUBSCRIPTION TERMS: \$6.00 per year in advance, postage paid for the United States, \$7.50 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 126 Massachusetts Ave.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.

### THE CHIROPRACTIC BILL

THE progress of this bill through the Legislature brought it before the House Ways and Means Committee on April 12. The lawyer in charge of the hearing for the advocates of the bill introduced several members of the Legislature who spoke in favor. He closed the hearing for the proponents in a long and somewhat impassioned plea for those people who want to have this form of treatment. He predicted the final adoption of this legislation. The arguments were based principally on alleged cures of various maladies after unsuccessful treatment by physicians and the claim that since Chiropractors do not practice surgery or obstetrics at the time of parturition the time devoted to the study of these subjects was wasted. He contended that if Chiropractors were obliged to qualify according to present methods in the state, they would be like present-day Osteopaths, less interested in the Chiropractic technic with the result that this alleged valuable form of treatment would be less frequently used and thereby lost to those who may need it most. This part of his argument was amusing to say the least. He particularly emphasized the belief that the opposition

to this bill is founded on the disposition on the part of doctors to prevent competition.

Dr. J. S. Stone opened the arguments in opposition to the bill calling attention to inaccuracies in the statements of the counsel for the Chiropractors and made a strong plea for the maintenance of the single standard of requirements now in the law relating to the practice of medicine. Several members of the Legislature and osteopaths endorsed the arguments of Dr. Stone. The President of the Osteopathic Society pointed out that if the Chiropractors are to be given a special examining board, the osteopaths would also demand a separate board, and this would be followed by appeals for boards representing all the cults. This would establish many forms of class legislation. Several women also endorsed Dr. Stone's argument, among them being Miss Sally Johnson, President of the Massachusetts State Nurses Association, and Mrs. Colin W. Macdonald.

Dr. O'Brien, Secretary of the Committee on Legislation presented letters from the presidents of colleges in Massachusetts and others all in favor of the single standard.

Dr. Charles E. Abbott of Andover, a former member of the House, made a logical and convincing argument in which he explained some of the inconsistencies of the proponents and especially refuted one of the letters which had been submitted by a Senator endorsing the bill. Two members of the Legislature who served on the recess committee which reported two years ago in favor of maintaining a single standard also submitted strong arguments against the bill.

The objection to the bill staged one of the most dignified and effective protests against this sort of legislation ever witnessed by the writer.

We must now wait for the report of the Committee and the action of the House.

Some members of the Committee seemed to realize the danger inherent in the passage of the bill. We hope that a sufficient number of objectors may be found who will endorse the arguments presented by the opponents.

### SOME PROBLEMS RELATING TO THE USE OF ALCOHOL

RECENTLY a statement in the press purported to quote Dr. George Bigelow, Commissioner of Health of Massachusetts, that there had been in recent years an increase in the number of deaths in this state due to the use of alcoholic beverages.

The Statistical Bulletin of the Metropolitan Life Insurance Company for March, 1927, presents the conclusion that since 1920 this increase has been very general. Among the Metropolitan Life Insurance industrial policy holders, the combined death rate for alcoholism and acute

alcoholic poisoning in 1926 was 4.1 per 100,000 which is the same rate as in 1911. The 1926 death rate was the highest since 1917 and is an increase of twenty-four per cent over that for 1925 and was three and one-sixth times that for 1920, which was the year in which the minimum death rate from alcoholism was recorded.

During the war years in England and Ireland, large numbers of people abstained from the use of alcohol. The same conditions existed in the United States in 1918.

The statistics of the Metropolitan Life Insurance Company show that among the industrial policy holders, the years 1912, 1913, and 1916 recorded the maximum death rate from this cause of 5.4 per 100,000, but in 1920 in the same group the rate was 1.3. The figures for 1926 shown above indicate an increase which if continued will bring the rate for 1928 to figures larger than for any previous year in the series referred to. Maryland and New York show the highest rates of 10.1 and 6.3 per hundred thousand for 1926 respectively.

The deaths due to acute poisoning by "wood or denatured alcohol" were relatively common in New York, New Jersey, Pennsylvania, Illinois, Massachusetts, Maryland, Ohio, and Tennessee. It is suspected that the statistics would be augmented if more careful diagnoses were made, but so far as known the mortality from alcoholic beverages containing wood alcohol or other poisonous combinations has decreased since 1920. That is, while there is an increase in deaths reported as due to alcoholism, there has been a decrease in the deaths reported as acute alcoholic poisoning.

Information acquired through this study shows that the average age of deaths among males due to alcoholism is practically stationary, but for females the average age period is lower. This raises the question as to the use of alcohol by young women. These figures are based on studies of conditions found in mortality returns of thirteen of the larger states and seem to be a fair index of conditions throughout the country. As statistics they show the great importance of the conditions which affect the health and economic problems before the country.

There is abundant evidence of unrest in the minds of the people at large in this country with respect to the whole problem of prohibition and the therapeutic value of alcohol, and there seems to be a very definite rebellion in the medical profession against the restrictions imposed in the regulations governing the medicinal use of alcohol. Public debates and space in the press devoted to consideration of morals, health and economic features of the enforcement of violation of the Volstead Act show that radical differences of opinion exist throughout the different strata of society even reaching to the Chamber of the Supreme Court of the United States. Since the majority of physicians represent the

intelligent portion of the population and are concerned especially in all health problems, there seems to be laid upon the profession a weighty responsibility demanding study of the subject in order to exert influence which may help to bring order out of the almost chaotic conditions now existing.

The first dictionary definition of the word "doctor" is that of "teacher." Our foremost doctors are devoting much time to education. Is it too much to expect that the profession will take on the function of teacher in the ethical and health problems of the use and abuse of alcohol in its relation to the well being of the race even though this may lead to more or less political activity?

#### THIS WEEK'S ISSUE

CONTAINS articles by the following authors:

BIRNIE, JOHN M., A.B.; M.D. Harvard Medical School 1906, F.A.C.S.; Member of the New England Surgical Society; Visiting Surgeon Springfield Hospital; Member of the State Board Registration in Medicine; Vice-President Massachusetts Medical Society. His subject is: "The Diagnosis of Abdominal Emergencies." Page 635. Address: 14 Chestnut Street, Springfield, Mass.

FRIED, B. M., M.D. University Medical School, Saratow, Russia; Voluntary Graduate Assistant in Dr. Cushing's service at the Peter Bent Brigham Hospital; Graduate Assistant in the Medical Clinic of the O. P. D. Massachusetts General Hospital. Formerly Assistant to Prof. Besredka. Pasteur Institute, Paris, and to Prof. S. B. Wolbach, Harvard Medical School and the Peter Bent Brigham Hospital. His subject is: "Some Clinical Aspects of Primary Carcinoma of the Pancreas." Page 640. Address: Peter Bent Brigham Hospital, Boston, Mass.

WHITE LEON E., A.B.; M.D. Dartmouth Medical School 1893; Otolaryngologist Massachusetts Eye and Ear Infirmary; Otolologist Massachusetts General Hospital; Member of the New England Otolaryngological Society, the American Academy Ophthalmology and Otolaryngology, the American Laryngologists, Rhinological and Otological Society, etc. The subject of his paper is: "A Plea for Conservatism in the Treatment of Optic Nerve Disturbances from Focal Infection." Page 644. Address: 390 Commonwealth Avenue, Boston, Mass.

SMITH, MILLARD, B.S.; M.S.; M.D. Harvard Medical School 1923; Assistant in Thorndike Memorial Laboratory, Boston City Hospital. His subject is: "The Use of Diets With Very Low Protein Content in the Treatment of Chronic Nephritis with Nitrogen Retention." Page 649. Address: 472 Commonwealth Avenue, Boston, Mass.

TALBOT, FRITZ B., A.B.; M.D. Harvard Medical School 1905. Clinical Professor Pediatrics Harvard Medical School; Chief Children's Medical Department, Massachusetts General Hospital. His subject is: "Directory for Wet-Nurses." Page 653. Address 270 Commonwealth Avenue, Boston, Mass.

## LEGISLATIVE NOTES

HOUSE BILL 1104 relating to the distribution and sale of certain dangerous caustic or corrosive substances was amended as it appeared in Senate bill 287 and was finally passed and became a law on April 6, 1927.

Dr. Francis P. Emerson was the original author of this bill and is entitled to the credit of securing favorable action by the legislature.

We are informed that an amendment of the Chiropractic bill is being considered by the House Committee on Ways and Means which provides that those who have practiced chiropractic during the past three years will come before the Board of Registration and Medicine and be examined in the subjects specified by the Board, and that hereafter the regular educational provisions of the bill will become operative if the bill passes.

## MISCELLANY

### THE NEW YORK CANCER ASSOCIATION ACQUIRES A HOSPITAL

HUDSON TOWERS, a twenty-seven story hospital building, now under construction at Seventy-seventh Street and West End Avenue, will be opened this summer as a National Cancer Center by the New York Cancer Association.

Four hundred beds will be available, either free or at a low cost to patients. A fund of five million dollars is to be raised, three and a half million to be used in paying for the hospital and nearly half a million to be used in procuring six grams of radium.

A clinic for five hundred patients will be operated. Research laboratories and an educational department will be features of the work carried on.

### SAVING CHILDREN FROM DIPHTHERIA

NEW HAVEN, Conn., seems to have found a pleasant and effective way to spread the gospel of diphtheria prevention. The health officer

sends to each baby in the city on his first birthday a pretty birthday card, with a letter to the parents calling attention to the importance of protecting the child against diphtheria. The city reported but one death from the disease during 1926.

In this connection, the Metropolitan Life Insurance Company reports for the year 1926 a new low death rate for diphtheria among its policyholders. It asserts that there is no good reason why the mortality from this scourge of childhood should not continue to decrease until it becomes negligible.—*Bulletin U. S. Dept. of Labor.*

## CORRESPONDENCE

### AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,  
March 28, 1927.

In addition to the articles enumerated in our letter of February 26, the following have been accepted:

#### Abbott Laboratories

Abbott's Mineral Oil Emulsion.  
Ephedrine Hydrochloride—Abbott.

#### Eli Lilly & Co.

Ephedrine Sulphate—Lilly.  
Pulvules Ephedrine Sulphate—Lilly, 0.025 gm.  
Pulvules Ephedrine Sulphate—Lilly, 0.05 gm.  
Ampoules Ephedrine Sulphate—Lilly, 1 cc., 0.05 gm.  
Solution Ephedrine Sulphate—Lilly, 3 per cent.

#### E. R. Squibb & Sons

Scarlet Fever Streptococcus Toxin—Squibb, 1 cc.

#### Towt-Nolan Laboratory

Lactobacillus Acidophilus Milk (Towt).

#### Nonproprietary Articles

Ephedrine.

Yours truly,

W. A. PUCKNER, *Secretary*,  
Council on Pharmacy and Chemistry.

### TUBERCULOSIS IN BOSTON AND ELSEWHERE

Recently there have appeared three reports dealing with tuberculosis in Boston: (1) By Murray Howard, (2) by C.-E. A. Winslow, (3) by Haven Emerson. Each of these has, and such reports apparently must have, a somewhat statistical basis. In two at least of these it is made to appear that Boston is in a very bad way with respect to tuberculosis; but I see little evidence of any real care in the statistical analysis.<sup>1</sup> It is even doubtful whether medical and

<sup>1</sup> It must be understood that I refer only to the statistical work; as to the value of the various conclusions or recommendations for action I am incompetent to judge, except that I cannot see how they either stand or fall on the basis of the statistical analysis and I conclude that they should be judged independently of it, as is often the case.

public health investigators appreciate the difficulties of making such an analysis as may be above the levels of drive or slander when used for the comparison of conditions in different districts. I cannot give an analysis such as I might desire, any more than they did or could, but, since the subject is so much discussed, it may be worth while for me to mix in the m  le enough to point out specifically the difficulties involved.

1. *The adjustment for non-resident deaths.* In Boston about 15% of deaths are those of non-residents. Some residents of Boston die elsewhere. How is the balance when struck? In Table 13 of the *U. S. Mortality Statistics, 1923*, it is shown that by this adjustment the death rate for Boston is reduced by 1.5 from 14.9 per M to 13.4, a matter of over 10%. For New York City there is no reduction, nor for Philadelphia. For Baltimore the reduction is from 15.0 to 14.0, or 7%. Unfortunately this analysis is not given by cause of death or by age and there is no apparent way to tell how much tuberculosis is affected by this adjustment; but as this is a disease in which there may be considerable migration we cannot be sure that rates for Boston and New York City might not be non-comparable unless an allowance of some 10% were made in favor of Boston.

2. *The adjustment for selectivity of population.* A part of this effect of migration may well not be recorded, because the migrants may have become actual residents of the place to which they went. For example, in 1920 according to *U. S. Mortality Rates, 1910-1920*, p. 74, the rate for tuberculosis of the lungs was highest in Colorado at 200.6 per CM and lowest at Utah at 33.5 or one-sixth as much. Does anybody believe that these figures indicate comparatively either natural climatic conditions or acquired public health activities favorable or unfavorable to death from tuberculosis? What would happen if, on reading these figures, all the tuberculous who could migrate should go to Utah that they might not die? Consider the figures (tuberculosis of the lungs) for mild Los Angeles as compared with rigorous Boston:

	Los Angeles		Boston	
	White	Colored	White	Colored
1922	158	338	87	411
1921	145	255	93	300
1920	155	350	104	378
Mean	153	314	95	363

Why is the white rate 60% higher for Los Angeles and the colored 14% lower than the rates for Boston?

3. *The adjustment for race.* As the illustration just given happens to show, the rate for the colored population is much higher than for the white. For this reason an adjustment must be made for color unless the figures are given for white and colored separately. But this is not all. Even in the white population there are marked racial differences which are discussed in *U. S. Mortality Rates, 1910-1920*, p. 74. In New York City, Philadelphia and Chicago, the rates for tuberculosis of the lungs for children of Irish mothers are about double the rates for all other whites and exceed them by about 100 per CM in the former two cities. It is presumable that the conditions are about the same in Boston. We have 23.4% of such in our white Boston population but only 10.8 in that of New York City. This should make a difference in our rate for whites of about 12 points. The rate for Jews is supposed to be low, and probably New York City has a far larger percentage of Jews than Boston and this should effect some lowering of the New York rate as compared with ours. It is not unlikely that on account of Jews and children of Irish mothers, 15 points might be allowed to Boston relative to New York. A complete adjustment for

race cannot easily be made, and in part must probably be estimated.<sup>1</sup>

4. *The adjustment for age.* Populations of different age distributions will not be comparable in respect to either tuberculosis or other rates unless allowance is made for the differences in age distribution. Fortunately many of the figures given by the Census are already age adjusted. For example, the crude rate for children of Irish mothers in New York City in 1920 was 242 but only 195 when age-adjusted; for children of native mothers it was 89 but increased to 94.3 by age adjustment.

5. *The allowances for differences in reporting or editing and for mistakes.* It is always difficult to estimate how much figures must differ before one is reasonably sure that the differences may not be due merely to diverse customs in reporting by physicians or in editorial classifications by official statisticians. For example, in New York City as a whole and in each of the boroughs of Manhattan and Brooklyn, which are tabulated by color, the rate for tuberculosis of the lungs in whites dropped from 1917 to 1918; in Boston, Baltimore and Philadelphia it rose. Then there is a curious course of tuberculosis statistics for the Bronx, where the rate for 1900 was 263 (perfectly normal for that time, Manhattan was 260), but jumped in 1901 to 527, from which it fell gradually to 411 in 1910 and then jumped back in 1911 to 169, from which, with minor fluctuations, it has fallen off to under 100 like other rates!!!

6. *The allowance for density.* According to Farr's Law, an infectious disease, and perhaps other types, should increase in incidence with an increase in the density of the population, partly perhaps from a diminution in the feasibility of hygienic living in crowded communities, partly from the increased infectious contacts that come with crowding. It is very difficult to estimate satisfactorily this allowance for density. The population of Boston is probably more dense than that of some large cities and less dense than that of others.

Factors other than these six could be mentioned, but I will proceed to an illustration, taking two boroughs, Manhattan and Brooklyn, of New York City, and Boston (*U. S. Mortality Statistics, 1922*, p. 40):

	Manhattan	Boston	Brooklyn
1922	118	104	80
1921	119	110	85
1920	140	122	105

These are age-adjusted rates for total populations and for all forms of tuberculosis. There is more difference between Manhattan and Brooklyn than between Boston and either. How matters might stand after due correction for the five factors other than age is not easy to say. The figures by color but otherwise unadjusted are:

Whites			
	Manhattan	Boston	Brooklyn
1922	97	87	69
1921	100	93	73
1920	120	104	93
Colored			
1922	327	411	237
1921	279	300	238
1920	330	378	339

<sup>1</sup> *U. S. Mortality Statistics, 1921*, pp. 109-117, gives an extensive study of the effect of race on the mortality rates of Boston and in particular cites 60 as the rate of tuberculosis for children of native mothers and 170 as that for children of Irish mothers, both for 1920. Those rates are exclusive of non-residents who died in Boston and of residents who died outside of Boston and hence are non-comparable with the figures 89 and 242 respectively for New York City or 77 and 201 for Philadelphia, but the difficulty is to get them up so high with proper allowances!



Our white rates again lie between those of the two boroughs; but our colored rates are higher.

The colored rate is high. That is true for "all causes" of death.

*Colored—All Causes*

	Manhattan	Boston	Brooklyn
1922	1809	2193	1941
1921	1732	1914	1826
1920	1918	2530	2148

To what the high general rate may be due I cannot say—possibly in part to the rigorous climate, possibly in part to the age distribution (the rates for cancer and heart disease among the colored in Boston are both a good deal higher than in Manhattan or Brooklyn). Boston's death rates among the colored need careful study; the tuberculosis rate fell off very little in the 13 years 1910 to 1922, while the rates in some other cities were showing a considerable decline.

It is of course unsound to base inferences either on inadequately adjusted data or on short series of data whereupon statistical fluctuations of a more or less accidental nature may operate. For example, from 1914 to 1922 the tuberculosis rate in Manhattan among whites fell from 199 to 97, a decrease of 102 points or 13 points per annum; but from 1910 to 1922 it fell only from 166 to 97, a decrease of only 69 points or 6 points per annum (less than half as much). On the other hand, from 1914 to 1922 the Boston rate for whites fell from 134 to 87, a decrease of 47 points or 6 points per annum, and from 1910 to 1922 the fall was from 166 to 87, a decrease of 79 points or 7 points per annum. To get any comparable figures on rates careful computation of the trends on properly adjusted figures must be made.

Taking the whole recent course of pulmonary tuberculosis in the cities of over 500,000 population into account and considering the allowances which might have to be made for various major factors bearing on the situation, I can see no reason to feel that Boston is, as compared with other cities, in the dreadful and disgraceful statistical position with respect to this disease that some persons would have us believe; but I do most sincerely wish that an adequate amount of money and an adequately trained man were available to get at the real facts. I have merely tried to put together some of the facts that for some time have been available to all in the Government reports.<sup>2</sup>

EDWIN B. WILSON.

<sup>2</sup> If we work with crude rates and the percentage of population which is of Irish mothers we seem to get for Boston rates both for these and for all other whites for 1920 which are well below those for either New York City or Philadelphia. I should guess at an Irish rate of 185 and a white rate of 86 for Boston to compare with the figures given for the other two cities. With the presumptive correction for density (Farr's Law) this would make the rates in all three cities practically alike in 1920, as they are without any corrections, and as one might expect them to be. The method of analysis is as follows: For cent. children of Irish mothers, Boston 23.4, New York City 10.8, Philadelphia 12.3. Crude rate for pulmonary tuberculosis in whites, Boston 194, New York City 106, Philadelphia 106. Rate for children of Irish mothers, New York City 242, Philadelphia 201. Consequent rate for all other whites, New York City 90, Philadelphia 85. Let  $x$  be the rate in Boston for the former, and  $y$  that for all other whites. Then the equation  $23.4x + .766y = 104$  represents our knowledge. If  $y = 70$ , then  $x = 211$ . Such a pair of values is possible and not inconsistent with the 60 and 176 given in Note 2. If  $y = 80$ , then  $x = 185$ , and these values are not inconsistent, either. The difference between crude and age-adjusted rates is considerable for New York and Philadelphia: All white (N. Y.) 99 instead of 116, (Phil.) 97 instead of 106; Irish (N. Y.) 195 instead of 242, (Phil.) 173 instead of 201; all others (N. Y.) 88 instead of 90, (Phil.) 85 instead of 93 (the order of the two cities has been interchanged by a differential gain of 5 for Phil. as compared with N. Y.). We do not have the all white adjusted rate for Boston and cannot proceed.

THE HOSPITAL CLINICAL CONGRESS OF  
NORTH AMERICA

NEWS BULLETIN No. 1

With Dr. M. T. MacEachern, director of the American College of Surgeons, I have been making a swing

around the entire North American continent conducting sectional hospital meetings.

The outstanding feature of the meetings is the new way in which Dr. MacEachern is carrying on the Hospital Conferences.

They have been conducted after the manner of clinics—the equipment, the organization and the procedure being set before the audiences by people selected from hospitals where the sectional meetings are being held.

The direct result was an increased interest in the equipment, the organization and the local procedure. Most interesting questions developed in the round-table discussions.

It is safe to say that much interest has been aroused among hospital people for hospital equipment, and the uses and the procedure connected with the equipment.

Our trips so far have covered the East, Southeast, South and Southwest, and in April and May we hope to complete the circuit of the entire United States and Canada.

The Hospital Clinical Congress of North America is a national effort to put the whole hospital layout in each of its departments before the hospital world.

Dr. MacEachern is director general of clinics for the congress.

The success of the sectional clinical gatherings presages the interest which will be evidenced in the congress being held in Milwaukee. In the sectional gatherings I brought up the plan for the congress and it has received an enthusiastic welcome with promises of support from all hospital people.

REV. C. B. MOULNIER, S.J.

AN ATTEMPT TO JUSTIFY HIS PRACTICE

March 29, 1927.

Editor, Boston Medical and Surgical Journal:

May I be permitted a little space in the JOURNAL respecting a notice sent you by the secretary of the Board of Registration in Medicine and published in March 24th issue, page 503?

According to his published letter it seems that the vote for revoking my registration was taken one week before the date set for a hearing which was on March 10. At the hearing the complaint alleged an illegal operation on January 28, while the testimony of facts in the case, by the patient herself, clearly showed that she, a 40-odd-year-old married woman, had been having heavy uterine hemorrhages for several days prior to the curettement I undertook on January 28, and that she had been attended during that time by a trained nurse who assisted at the operation.

To do anything "illegal" was farthest from my thoughts and as no foetus was found it was questionable to me if this was more than a uterine polyp.

Respectfully,

FRANK S. PARSONS, M.D.

A MEDICAL REVIEW OF THE SIXTY-NINTH  
CONGRESS

(From Our Special Correspondent)

Although the Sixty-ninth Congress has taken its place, such as it is, in history, a résumé of the action by this Congress on matters of direct interest to physicians may be of interest and value.

Out of the 25,000 bills and resolutions introduced in this Congress, the first session of which convened in December, 1925, and the second session of which ended on March 4, 1927, only about 150 were of direct interest to physicians and sanitarians. Out of about 1,800 new laws, of which more than half were concerned with private matters, only four of any real significance to national vitality were enacted. Of

these four, one was primarily of more economic than sanitary import; one dealt with an institution in Panama; and one acts virtually to repeal a health law, the Maternity and Infancy Act, though many persons are convinced that this is a positive achievement. The failure of certain other measures which would have exerted a baneful effect on medical affairs is also a favorable accomplishment.

#### NEW HEALTH LAWS

The four important new health laws are: The act to safeguard the distribution and sale of dangerous caustic and corrosive substances in interstate commerce; the act for the two-year extension of the Federal Maternity and Infancy Act; the act for the regulation of imported milk; and an act authorizing an appropriation to the Gorgas Memorial Institute of Tropical Medicine at Panama. Of less general significance were acts to continue hospitalization of certain veterans at Liberty, N. Y., and at Saranac, N. Y.; an act for payments for blood transfusions in government hospitals; an act authorizing payment of expenses of delegates to a Pan-American sanitary Conference at Lima, Peru; an act relative to fees of surgeons in Alaska, which actually concerns only one person; and an act for narcotic distribution in the Virgin Islands.

Another matter of importance which should be mentioned is the Revenue Act of 1926, in which the tax on physicians under the narcotic laws was reduced from \$3 to \$1 a year. A bill to "strengthen" the Harrison Narcotic Act, which would have imposed new and severe restrictions on physicians, was fortunately not acted upon. A dangerous bill relating to medical practice in the District of Columbia, which would have opened the doors wide and invitingly to the cults, also did not get to a vote. No progress was made on the usual anti-vivisection measure.

The failure of these obnoxious bills is, of course, gratifying, but a large number of excellent measures likewise failed to receive the attention they merited. Many of these passed the House, but were defeated by the time-wasting filibuster in the Senate. Thus, the plan for the manufacture of medicinal liquor was not taken up in the Senate, though adopted in the House, and a similar fate was met by the proposal for additional hospital facilities for veterans.

#### HEALTH BILLS NOT PASSED

Among the bills upon which no final action was taken were those for correlation of Federal health activities; for a national institute of health; for Federal subsidies for rural health work; authorizing a five million dollar reward for a cancer cure; for the national control of narcotics; for a survey of Indian conditions; for granting a commissioned status to the sanitary engineers of the Public Health Service; authorizing this Service to give special advice on sanitary engineering; control of oil pollution of navigable streams; granting a commission as colonel to the President's physician; for the regulation of osteopathy in the District of Columbia; for workmen's compensation for longshoremen, including medical benefits; for the repeal of the act creating the Children's Bureau; for a system of school hygiene in the District of Columbia; for the testing of clinical thermometers; and numerous bills relating to the hospitalization of veterans in special places, and many measures proposing amendments to the pure food laws.

Funds for the operation of the Federal Maternity Act as extended were tacked on to the general appropriation bill at the last moment, so that this activity was not curtailed by the failure of the passage of the deficiency appropriation bill.

#### THE FEDERAL CAUSTIC POISON ACT

The new law (Public No. 783, Sixty-ninth Con

gress) to safeguard the distribution and sale of certain dangerous caustic or corrosive acids, alkalis, and other substances in interstate and foreign commerce represents a real achievement by the organized medical profession, for this measure was prepared and sponsored by the American Medical Association. The law requires adequate labelling with the word "Poison" of cans of lye and similar substances, with directions for emergency treatment. The penalty is not to be enforced for six months after the passage of the act, which was signed by the President on March 3. Laws regulating caustics are said to be also now in force in fourteen States.

#### COMMENT

Detailed description of the other new health laws need not be given, as they have been frequently commented upon in these letters. Medical and public health matters did not fare particularly well at the hands of this Congress, and that is the usual story with every Congress. The care of the public health has been said to be the first duty of the statesman, but this axiom does not seem to be taken seriously by our national legislators. It probably never will be until the medical and sanitary professions make a sufficiently vigorous impression on their chosen representatives concerning the importance of the promotion of national vitality, and are supported in this laudable endeavor by the greater part of the intelligent lay public.

JAMES A. TOBEY.

#### NEWS ITEM

**SURGEON GENERAL CUMMING WILL GO TO HONOLULU AFTER ATTENDING CONFERENCE IN CHICAGO**—Dr. Cumming addressed a conference at Chicago in session March 24 and 25 between official health agencies of the United States and the American Medical Association. He will thence proceed to Honolulu to attend the Pan-Pacific Conference which will assemble for the discussion of problems of mutual interest to countries represented, relating to the betterment of public health, education and commerce.

During his absence Dr. C. C. Pierce will be acting surgeon general.

#### NOTICES

**DR. TRACY JACKSON PUTNAM** announces the opening of an office at 270 Commonwealth Avenue, Boston, Massachusetts, in association with Dr. Gilbert Horrax.

**TO THE EDITORIAL STAFF OF THE BOSTON MEDICAL AND SURGICAL JOURNAL, BOSTON**

Gentlemen:

Please publish the following notice in the editorial column of your esteemed journal, which we feel sure will be of vital interest to many of your readers.

*International Medical Postgraduate Courses* will be held in Berlin by the Union of Professors and by the organization centered in Kaiserin Friedrich-Institute with the assistance of the medical faculty of the university. One section of the courses will be held continuously, another section only during the period 15th September to 31st October.

#### I. Continuous Courses:

(a) Individual courses in all branches of medicine with practical exercises usually lasting four weeks.

(b) "Externships" in clinics and hospitals and laboratories. These are chiefly intended for medical men desirous of working under practical conditions

for a longer period (at least two to three months).  
*II. Courses from 15th September to 31st October.*  
In the autumn it is intended to hold:

(a) General courses in all the branches of internal medicine with special regard to modern therapy (duration 14 days).

(b) General courses in stomach and intestinal diseases.

(c) General courses in pediatrics.

(d) General courses in gynecology.

(e) Special courses in medical technique at the sick-bed and in the laboratory with practical exercises and demonstrations.

(f) Special courses for nose, ear and throat specialists.

(g) Individual courses in all departments of medicine with practical participation as given in a specially prepared list.

The medium of instruction is German, but there are a number of lecturers who are able to instruct in the English, French or Spanish languages.

The Bureau would be very glad to furnish addresses where suitable accommodation is available, and to give information in regard to cost of living, etc.; also to arrange facilities for seeing operations in clinics and similar services.

Every medical man who intends to visit Germany or particularly Berlin for the purpose of continuing his studies is advised to get into touch with the Bureau, which has an official character.

The address of the Bureau is:

Kaiserin Friedrich-Haus,  
Nos. 2-4 Luisenplatz,  
Berlin NW 6.

#### UNITED STATES CIVIL SERVICE EXAMINATION

The United States Civil Service Commission announces the following open competitive examination:

Associate Bacteriologist (Medical)

Applications for associate bacteriologist (medical) must be on file with the Civil Service Commission at Washington, D. C., not later than May 10.

The examination is to fill a vacancy in the Hygienic Laboratory of the Public Health Service, Washington, D. C., at \$3,600 a year, and vacancies occurring in positions requiring similar qualifications, at approximately the same rate of pay.

The duties are to perform independently or with associates or through subordinates, work of major importance in bacteriology.

Competitors will not be required to report for examination at any place, but will be rated on their education, training and experience; and a thesis or publications on some subject connected with bacteriology, to be filed with the application.

Full information and application blanks may be obtained from the United States Civil Service Commission, Washington, D. C., or the secretary of the Board of United States Civil Service Examiners at the postoffice or custom house in any city.

#### UNITED STATES PUBLIC HEALTH SERVICE

##### CHRONOLOGICAL LIST OF CHANGES OF DUTIES AND STATIONS OF COMMISSIONED AND OTHER OFFICERS OF THE UNITED STATES PUBLIC HEALTH SERVICE

MARCH 30, 1927

Acting Assistant Surgeon John C. Travers—Directed to proceed from Baltimore, Md., to New York City, and return, to testify in court in behalf of the United States Shipping Board Emergency Fleet Corporation—March 11, 1927.

Surgeon W. C. Teufel—Relieved from duty at Stockholm, Sweden, and assigned to duty at Berlin, Germany—March 21, 1927.

Surgeon J. P. Leake—Directed to proceed from Washington, D. C., to New York City, and return, in connection with tetraethyl lead gasoline investigations—March 22, 1927.

Associate Sanitary Engineer A. P. Miller—Directed to proceed from Washington, D. C., March 22, to New York City, and return, for conference regarding Service activities—March 22, 1927.

Statistician Edgar Sydenstricker—Directed to proceed from Washington, D. C., to Baltimore, Md., April 1, and return, in connection with the study of vital statistics methods of the Maryland State Health Department—March 23, 1927.

Senior Sanitary Engineer J. A. Leprince—Directed to proceed from Memphis, Tenn., to Jackson, Miss., and return, to attend a meeting of the Southeastern Water and Light Association to be held in that city April 26-28—March 24, 1927.

Associate Sanitary Engineer Leonard Greenburg—Directed to proceed from New Haven, Conn., to Washington, D. C., and Wilmington, Del., and return, in connection with ventilation studies—March 24, 1927.

Assistant Surgeon Albert E. Russell—Directed to proceed from Johnstown, Pa., to Washington, D. C., and return, in connection with dust studies—March 24, 1927.

Acting Assistant Surgeon George B. Story—Directed to proceed from Tacoma, Wash., to Seattle, Wash., for assignment to duty at U. S. P. H. S. Relief Station No. 329—March 24, 1927.

Surgeon R. E. Dyer—Directed to proceed from Washington, D. C., to Scranton, Pa., March 25, and return, in connection with the control of biologic products—March 25, 1927.

Surgeon T. J. Liddell—Directed to proceed from Stockholm, Sweden, to points in Continental Europe from time to time during the present fiscal year as may be necessary, and return, in connection with Service activities—March 26, 1927.

Assistant Surgeon W. H. Sobrell—Relieved from duty at Stapleton, N. Y., and assigned to duty at United States Quarantine Station, Rosebank, S. I., N. Y.—March 26, 1927.

Assistant Surgeon F. C. Stewart—Relieved from duty at Rosebank, S. I., N. Y., and assigned to duty at United States Quarantine Station, Angel Island, Calif.—March 26, 1927.

Special Consultant Walter M. Brunet—Directed to proceed from New York City to Syracuse and Olean, N. Y., and Cleveland, Ohio, and return, in connection with studies of venereal diseases—March 26, 1927.

Acting Assistant Surgeon Neil E. Taylor—Bureau orders of March 22, 1927, directing him to proceed from Cincinnati, Ohio, to Portland, Oregon, and other places in that State, in connection with field investigations of gonorrhea, revoked—March 26, 1927.

Surgeon L. D. Fricks—Directed to proceed from Seattle, Wash., to Winthrop, Wash., and return, to investigate a claim for the United States Employees' Compensation Commission—March 28, 1927.

#### BOARDS CONVENE

A board of officers convened to meet at Philadelphia, Pa., at the call of the chairman, for the purpose of reexamining an alien—March 25, 1927. Detail for the board: Surgeon H. E. Trimble, A. A. Surgeon Leon Van Horn, A. A. Surgeon Horace Phillips.

A board of officers convened to meet at Seattle, Wash., April 11, 1927, to determine the physical eligibility of an officer of the United States Coast Guard for promotion to permanent commander—March 26, 1927. Detail for the board: Surgeon L. D. Fricks, Surgeon Carl Michel.

Official:

C. C. PIERCE, Acting Surgeon General.

## PRIZE WINNING HEALTH PLAYS

THE third annual production, Massachusetts prize winning health plays of three high schools of the Commonwealth in the National Health Playwriting Contest, will be staged at the Copley Theatre, Boston, Friday, April 29th, at 3:30 P. M.

High school pupils, teachers, parents, physicians, nurses, social workers and the public cordially invited. Tickets only thirty-five cents. Massachusetts Tuberculosis League, 1149 Little Building, Boston, Mass.

## RECENT DEATH

**TUTTLE**—DR. GEORGE THOMAS TUTTLE died on April 6, 1927, at his home, 110 Highland Street, Milton, at the age of 77 years, from pneumonia.

He was born at Northwood, N. H., March 18, 1850. He was a graduate of Dartmouth College, class of 1872, and of the Harvard Medical School in 1878. After a medical house-officership at the Boston City Hospital he was for a short time assistant superintendent there, and then on April 15, 1879, he began a 40-year service at McLean Hospital, which he terminated by resignation on April 15, 1919. During practically 25 years he was first assistant physician, and for 15 years superintendent. On retiring, he was elected to the board of trustees of the Massachusetts General Hospital. In 1908 he was appointed by the Governor chairman of a commission to revise and codify the laws relating to the insane. The recommendations made by the commission are the basis of our present laws. He was president of the Middlesex South District Medical Society in 1917 and 1918, and, during the war, chairman of the local Auxiliary Medical Defense Committee. From 1905 to 1912 he was clinical instructor in psychiatry at the Harvard Medical School. He was a member of the Boston Society of Psychiatry and Neurology, of the Massachusetts Psychiatric Society, of the New England Society of Psychiatry, and the American Psychiatric Association, as well as of the Massachusetts Medical Society.

## OBITUARY

## DR. ARTHUR B. WETHERELL

ON March 18th Dr. Arthur B. Wetherell died at his home in Holyoke of coronary thrombosis. For two years he had been in ill health, but had continued in the active practice of his profession until he was fatally stricken.

Since 1886 Dr. Wetherell had been active in the medical profession in Holyoke, being a member of the board of health for about 15 years, an active member of the staff of the Holyoke City Hospital for many years, and one of the founders of the Tuberculosis Hospital. He was also one of the mill physicians for a great many years.

Dr. Wetherell was born in Southampton February 2, 1855, the son of Barney and Adelia (Stedman) Wetherell. He received his early

education in the schools of that town and later attended Williston Seminary. He later attended Harvard Medical School, graduating in 1883. During the next two years he was connected with Boston hospitals, gaining wide experience through this service.

On April 6, 1886, he came to Holyoke and remained for about 41 years one of the most active as well as one of the most prominent physicians in this section. He was one of the most kindly of men and as a physician he frequently served his fellowmen without thought of recompense. There was general sorrow among his large circle of friends when his death became known.

He was instrumental in the organization of the Holyoke City Hospital; was associated with Dr. L. M. Tuttle in selecting the site of the hospital, and for many years was a member of the hospital board. In the late nineties he was selected as a member of the board of health and served faithfully on that board until 1912, when he retired.

Dr. Wetherell was a member of the Holyoke Medical Society, the Massachusetts Medical Society, the American Medical Association, and the Harvard Medical Alumni. He was affiliated with the Masonic Order, being a member of the Mt. Tom Lodge, Mount Holyoke Chapter, Royal Arch Masons, Holyoke Council, Royal and Select Masters, Springfield Commandery, Knights Templar, and the Ancient Arabic Order Nobles of the Mystic Shrine. He was also a member of Unity Lodge, Independent Order of Odd Fellows; Knights of Pythias, Mount Tom Golf Club, and the Holyoke Canoe Club. He had been forced to relinquish his membership in some of these societies because of ill health.

During his term on the Board of Health, Dr. Wetherell, with Dr. Frank A. Woods, an associate, launched the Tuberculosis Hospital project, which he lived to see develop. He also served as a member of the Board of Pension Examiners of Northampton.

Until the early part of last year he had maintained an office at the corner of Chestnut and Appleton Streets and was very active in the practice of his profession. Since that time he discontinued his downtown office but continued to receive patients at his home on Fairfield Avenue until the end.

Dr. Wetherell was married in 1886 and Mrs. Wetherell survives him with one son, Dr. Bryant David Wetherell of Boston, and one daughter, Mrs. Lawrence Davis of Cleveland. He leaves one grandson, Bryant David Wetherell, and three sisters, Mrs. Henry McLean of Ridgewood, N. J.; Mrs. William Smith of Waverley, and Miss Helen Wetherell of Northampton.



## REPORTS AND NOTICES OF MEETINGS

### CONNECTICUT CONFERENCE OF SOCIAL WORK

THE seventeenth annual session of the Connecticut Conference of Social Work will be held at Hotel Bond, Hartford, April 24-27, 1927.

The program presents names of national repute and offers so many discussions of home and community problems as to make it important for people to attend each and all of the sessions during that time.

### MEETING OF THE RECONSTRUCTION CLINIC

THE regular monthly meeting of the Physiotherapy Department of the Reconstruction Clinic will be held on April 28, 1927, at 8:00 P. M. Actino-therapy and its relation to general practice of Medicine, with practical demonstrations and case discussion.

All physicians interested are cordially invited.

### ANNUAL MEETING OF THE BERKSHIRE DISTRICT MEDICAL SOCIETY

PLACE: New American House. Time: Thursday, April 21, 1927. Dinner at 6:30 P. M. Meeting directly after dinner. Business: Election of officers. Speaker: Abraham Myerson, M.D., of Boston. Subject: "Significance of Spinal Fluid Findings in Disease."

### THE ANNUAL MEETING OF THE MASSACHUSETTS TUBERCULOSIS LEAGUE

DR. JOHN A. KIERNAN, Chief of the Division of Tuberculosis of the United States Bureau of Animal Industry, will be the principal speaker at the thirteenth Annual Meeting of the Massachusetts Tuberculosis League which will be held this year at the Hotel Statler, Boston, on Tuesday, April 26th. Dr. Kiernan will address the meeting on "The Eradication of Bovine Tuberculosis in the United States." Dr. James S. Stone, President of the State Medical Society, will speak on "The Progress of Tuberculosis Work." Election of officers will follow the luncheon program.

Dr. Kendall Emerson, President of the League, will preside at this Meeting and will give a short address on the progress of the work to eradicate tuberculosis in Massachusetts. Those invited to attend the affair are the representatives of the twenty-seven affiliated organizations of the League, Federal and State medical and social workers, physicians, nurses, teachers and public health workers from all of the New England States.

Frank Kiernan, Executive Secretary, is in charge of all arrangements.

### UNION HOSPITAL IN FALL RIVER

THE regular monthly clinical staff meeting will be held at the Stevens Clinic on Thursday, April 21, 1927, at 8:15 P. M.

Paper by Dr. F. J. Sullivan of Fall River, Mass. Subject: "Dentistry and Its Relation to Medicine."

M. N. TENNIS, M.D., Secretary.

### HARVARD MEDICAL SOCIETY

THE next regular meeting of the Harvard Medical Society will be held on April 26 at 8:15 in the Amphitheater of the Peter Bent Brigham Hospital.

The program will be as follows:

1. Presentation of cases.
2. Dr. Hugh Cairns on "The Life of a Medical Student in England."
3. Dr. William L. Aycock on "Milk-borne Epidemic of Polio-myelitis."

There will be lantern slides.

Medical students and physicians are cordially invited.

### MEETING OF THE CENSORS OF THE ESSEX SOUTH DISTRICT MEDICAL SOCIETY

THE Censors of the Essex South District Medical Society will meet for the examination of candidates at the Salem Hospital, at 3:30 o'clock on Thursday, May 5th, 1927.

RALPH E. STONE, M.D., Sec'y.

### AMERICAN NURSES ASSOCIATION

THE fifth convention of the New England Division, American Nurses Association, will be held at the Biltmore Hotel, Providence, R. I., April 27, 28, 29, 1927.

President, Sally Johnson, R.N., Massachusetts General Hospital, Boston, Mass.; Vice-President, Lucy C. Ayers, R.N., Woonsocket, R. I.; Secretary, Esther Dart, R.N., Stillman Infirmary, Cambridge, Mass.; Treasurer, Ednah A. Cameron, R.N., 8 North Street, Concord, N. H.

### THE WILLIAM THOMPSON SEDGWICK MEMORIAL LECTURE

DR. HAVEN EMERSON, Professor of Public Health Administration of Columbia University, College of Physicians and Surgeons, delivered the sixth William Thompson Sedgwick Memorial Lecture at Massachusetts Institute of Technology on Friday, April 8. Dr. Emerson's subject was "Public Health Diagnosis." A digest of the lecture follows:

Diagnosis in public health as in the practice of medicine deals with the patient, in this instance a community, rather than with a particular disease.



It is by his use of the science and art of diagnosis that the professional sanitarian is distinguished from the routine administrator of codes and laws for health protection.

The beginning of the practice of revealing the health or sickness status of a community in the modern sense was made in England by John Howard in 1777 through his report upon the state of prisons in England and Wales. Modern institutional sanitation dates from this study.

In 1842 Edwin Chadwick presented his famous report upon the sanitary condition of the laboring classes in England and Wales in which observations upon births, deaths, sickness and the relations of health to housing, industry, climate, personal habits, race, and sex, etc., are recorded in a way to determine the content of most of the subsequent efforts in this field.

This convincing demonstration of the wastage of human life from entirely preventable causes and the description of desirable public measures for correction of sanitary evils resulted in the creation of a central health administration which has been a model for many states and nations since then, and is represented now by the Ministry of Health.

The next community to be dealt with by a competent diagnostician was the Commonwealth of Massachusetts for which Lemuel Shattuck did much the same great service as Chadwick did for England. The minuteness and range of topics considered in the Shattuck report have rarely been equalled and the impression left upon the public of this portrayal of reasons for their increasing death rates was strong enough to bring about some years later the first State Board of Health, an example of good organization, with ample powers and reasonably adequate funds.

In 1864 came the first medically directed diagnosis, and this time of a city population. The City of New York received at the hands of the Citizens Association, through its Council of Hygiene and Public Health, a medical examination such as no similar group of people had ever had before.

The findings and recommendations came with such force and carried such overwhelming conviction that there was general approval for a municipal health department with powers and duties so well conceived that to this day it has served almost in its original form.

For more than forty years no new development in this field occurred. With the record of a national, a state, and a city survey of health and the resultant standard of organizations, operating to apply sanitary science to all types of communities, the practice of public health diagnosis fell into disuse, to be revived from an entirely new angle through the force of social science in the Pittsburgh Survey of 1907.

The problems of the steel and iron city, of politics, polluted water, brutality of toil, indif-

ference of wealth, all forged together in the cauldron of immigrant races, presented a challenge met now for the first time by a joint endeavor of many professions with representatives of the community itself.

Between 1907 and 1917 there came a revival of public health diagnosis, stimulated by the desire for better civic government, supported by philanthropic funds, and assisted by a very real rivalry among communities themselves for something more secure and productive in health service.

The surveys of Topeka, Kansas, and Springfield, Illinois, are examples of excellent diagnostic undertakings.

Of a different nature were the administrative studies carried out by the United States Public Health Service, primarily intended to give a basis for reconstruction of statute law and of health department organization. Diagnosis played but a small part in these investigations.

In the past nine years there have been more community case histories written than in all the years before.

Innovations in procedure, in content, in object have come thick and fast. All the cities of 40,000 and over have had at least one record made of the essentials of their health status and many have had repeated examinations to reveal progress and to prove the results of the treatment advised.

A public health diagnosis today is an established procedure requiring the widest searching into the whole structure and all the vital functions of a community. There is provided through the American Public Health Association for the use of any city or rural area the professional skill by which Mayors and Common Councils, Community Chests or Welfare Federations can obtain a plan of treatment for health purposes as definite and promising as that of the internist based upon physical and laboratory diagnosis of the individual patient.

We may promise ourselves at least as much benefit from annual public health diagnosis of the population in its entirety as we now know we can obtain from periodic health examinations of its members.

New resources for information are in process of development and in the future we shall see the records of current sickness, of the prevalence of non-fatal disease, used with as much effectiveness as were the bills of mortality by Chadwick nearly a hundred years ago.

In the experience of the practicing physician and the daily rounds of the visiting nurse are the intimate facts upon which future diagnosis will be based. While much has been learned from a study of preventable deaths, such indices of a community health hazards are too remote, too unresponsive to the forces of modern social organization, transportation, communication of

word and thought for the health officer of today.

Progress in the applied medical, and particularly the biological sciences, of the coming years as they focus upon the great uncompleted job of health protection awaits improvement in the accuracy and thoroughness of diagnostic criteria applied to the study of human society. The physician and the sanitarian will remain for all time fellow students of disease as it modifies the length, breadth and depth of human life, whether in the individual or in the social unit. Only by providing responsible diagnosis before applying treatment will the practitioner of public health earn the right to enter the brotherhood of the professions and share with the family physician the respect of the community.

# MASSACHUSETTS TUBERCULOSIS LEAGUE, INC.

April 16, 1927.

## CALL FOR ANNUAL MEETING

NOTICE is hereby given that the Annual Meeting of Massachusetts Tuberculosis League, Inc., will be held Tuesday, April 26, 1927, at 12:15 o'clock at Hotel Statler, Boston.

The business to be transacted at this meeting is the election of officers and Board of Directors for the coming year.

The Constitution of the Corporation provides that all members of the League shall have a right to be heard at all meetings and that each shall be entitled to one vote. This means that the voters at the Annual Meeting are individuals who have joined the organization during the year by the payment of a membership fee of \$5 or over and delegates from society members of the League. Each affiliated organization of the League is entitled to one delegate to represent it at this meeting.

Attached is the list of nominations for officers and directors of the League for the coming year, nominated according to Article VII of the By-Laws of the Corporation. Other nominations for any office or director at large may be made from the floor.

Respectfully yours,  
FRANK KIERNAN,  
Clerk of the Corporation.

April 16, 1927.

## REPORT OF NOMINATING COMMITTEE OF MASSACHUSETTS TUBERCULOSIS LEAGUE INC.

The Nominating Committee, having been duly appointed by the President, Dr. Kendall Emerson, to serve for the purpose of bringing nominations for the general officers and for the Board of Directors at Large for the Massachusetts Tuberculosis League for the ensuing year, submit the following list of nominations:

For Directors at Large: Mr. Sydney Ashe, Pittsfield; Dr. George H. Bigelow, Boston; Dr. Vincent Y.

Bowditch, Boston; Dr. Walter P. Bowers, Clinton; Dr. Robert Carpenter, North Adams; Dr. Henry D. Chadwick, Westfield; Dr. Francis G. Curtis, West Newton; Mr. Arthur Drinkwater, Cambridge; Dr. Kendall Emerson, Worcester; Dr. Charles B. Fuller, Waltham; Dr. Harold A. Gale, Winchester; Mr. William B. Geoghegan, New Bedford; Mr. William N. Goodell, Lowell; Mrs. John D. Henry, Boston; Dr. William O. Hewitt, Attleboro; Dr. George S. Hill, Boston; Professor Murray P. Horwood, Newton; Dr. Roger I. Lee, Brookline; Dr. Frederick T. Lord, Boston; Dr. Adam S. MacKnight, Attleboro; Dr. Edward O. Otis, Boston; Dr. Sumner H. Remick, Boston; Mr. John Ritchie, Malden; Representative George L. Richards, Malden; Miss Margaret Weir, Beverly.

For Honorary President: Dr. Edward O. Otis, Boston.

For President: Dr. Kendall Emerson, Worcester.

For Honorary Vice-Presidents: Dr. Vincent Y. Bowditch, Boston; Dr. George H. Bigelow, Boston; William Cardinal O'Connell, Boston.

For Vice-President: Dr. Frederick T. Lord, Boston.

For Treasurer: Mr. Arthur Drinkwater, Cambridge.

For Assistant Treasurer: Mr. Romney Spring, Boston.

In addition, the Nominating Committee nominates as Clerk of the Corporation for the ensuing year Mr. Frank Kiernan, the Executive Secretary of the League.

Respectfully submitted,

DR. HAROLD A. GALE, *Chairman*.

MRS. REGINALD HEBER WHITE.

MR. WILLIAM N. GOODELL,

MRS. CAROLYN M. ENGLER.

DR. HILBERT F. DAY.

April 16, 1927.

## LIST OF REPRESENTATIVE DIRECTORS NOMINATED BY AFFILIATED ORGANIZATIONS

Barnstable County Public Health Association: Dr. Harry S. Wagner, Pocasset.

Berkshire County Tuberculosis Association: Dr. P. J. Sullivan, Dalton.

Boston Tuberculosis Association: Dr. Cleaveland Floyd, 246 Marlboro Street, Boston; Dr. John B. Hawes, 2nd, 11 Marlboro Street, Boston; Mr. Edmund H. Talbot, 35 Congress Street, Boston; Mr. Raymond S. Wilkins, 53 State Street, Boston; Mr. Arthur V. Woodworth, 92 Mt. Vernon Street, Boston; Mrs. Reginald Heber White, 3 Revere Street, Jamaica Plain.

Bristol County Public Health Association: Mr. Charles H. Holmes, Acushnet.

Cambridge Anti-Tuberculosis Association: Dr. Hilbert F. Day, 45 Bay State Road, Boston; Mrs. Mabel Greeley Smith, 689 Massachusetts Avenue, Cambridge.

Chelsea Women's Public Safety Committee: Mrs. E. Frank Guild, 8 Crescent Avenue, Chelsea.

Essex County Health Association: Dr. Olin S. Pettigill, Essex County Sanatorium, Middleton; Dr. Walter G. Phippen, 31 Chestnut Street, Salem.

Fall River Anti-Tuberculosis Society: Mr. James F. Sullivan, Box 195, Fall River.

Franklin County Public Health Association: Rev. C. P. Wellman, Deerfield.

Hampden County Tuberculosis and Public Health Association: Mr. Walter S. Barr, West Springfield; Mr. Clifton S. Hobson, Palmer; Mr. Frederic Edwards, 145 State Street, Springfield.

Hampshire County Public Health Association: Mr. C. E. Hodgkins, 133 South Street, Northampton.

Haverhill Tuberculosis Association: Dr. I. J. Clarke, 112 Emerson Street, Haverhill.

Holyoke Society for the Prevention of Tuberculosis: Dr. Solomon L. Skvirsky, 108 Essex Street, Holyoke.

Lawrence Tuberculosis League: Mrs. Frances B. Mowry, 84 Knox Street, Lawrence.

Lowell Tuberculosis Council: Mr. Charles H. Hobson, 158 Middle Street, Lowell.

Lynn Tuberculosis League: Mrs. Carolyn M. Engler, 12 Deer Cove, Lynn.

Malden Anti-Tuberculosis Society: Mrs. George A. Ricker, 40 Hancock Street, Malden.

Nantucket Public Health Committee: Miss Clementine Platt, R.N., 69 Orange Street, Nantucket.

New Bedford Anti-Tuberculosis Association: Dr. John M. Wise, Box 947, New Bedford.

Newburyport Anti-Tuberculosis Association: Mrs. Allan R. Shepard, 233 High Street, Newburyport.

Newton Welfare Bureau: Mr. Arthur Kendrick, 45 Hunnewell Avenue, Newton.

Norfolk County Public Health Association: Dr. Francis P. Denny, 111 High Street, Brookline; Dr. Nahum R. Pillsbury, Norfolk County Hospital, S. Braintree.

Northern Worcester Public Health Association: Mrs. Edyth James, R.N., 20 Exchange Street, Leominster.

Plymouth County Health Association: Mrs. B. Milo Burke, 12 Doris Avenue, Brockton; Dr. J. Holbrook Shaw, 43 Court Street, Plymouth.

Salem Association for the Prevention of Tuberculosis: Dr. J. Frank Donaldson, 32 Lynde Street, Salem.

Southern Middlesex Health Association: Miss Josephine B. Colt, Concord; Mrs. Josephine White, 26 Sanborn Street, Reading.

Southern Worcester County Health Association: Dr. Roy J. Ward, 8 Bellevue Street, Worcester; Rev. J. F. McGillicuddy, North Brookfield; Dr. Gardner N. Cobb, 36 Pleasant Street, Worcester.

The Public Health Association of Southwestern Middlesex County: Dr. Edward H. Ellis, Winthrop Street, Marlboro.

#### THE MEETING OF THE NEW HAMPSHIRE SURGICAL CLUB

THE 29th semi-annual meeting of the New Hampshire Surgical Club will be held at Keene, N. H., May the 3rd, 1927. The meeting as usual will consist of a clinic in the forenoon and after lunch a literary scientific program. The Keene doctors have been delegated committee of arrangements with Dr. Ira Prouty chairman.

It is interesting to note that Dr. Ira Prouty of Keene was the first secretary-treasurer of the New Hampshire Surgical Club and the only living charter member. The New Hampshire Surgical Club was organized at Concord, N. H., June the 1st, 1896, by six outstanding New Hampshire surgeons, Wm. F. Smith of Hanover, E. F. McQuestion of Nashua, A. C. Heffinger of Portsmouth, Geo. D. Towne of Manchester, Robert Burne of Plymouth, and Ira Prouty of Keene. Dr. E. F. McQuestion was elected president, Dr. Ira Prouty secretary. From this notable beginning the New Hampshire Surgical Club has grown to an organization of nearly two hundred and about the names of these founders is written notable events in the history of New Hampshire medicine.

#### MASSACHUSETTS TUBERCULOSIS LEAGUE EXECUTIVE COMMITTEE

A MEETING of the Executive Committee of the State Tuberculosis League was held at Massachusetts Institute of Technology School of Public Health on April 8. There were present: Drs. Kendall Emerson, Frederick T. Lord, George H. Bigelow, Walter P. Bowers, Roger I. Lee, Henry D. Chadwick, Francis P. Denny, Adam S. MacKnight, Mr. Arthur Drinkwater, Professor Murray P. Horwood of the faculty of the School of Public Health of Massachusetts Institute of Technology, Miss Anna W. Johnson, Educational Secretary of the League, and Mr. Frank Kiernan, Executive Secretary of the League.

It was announced that the Annual Meeting of the League will be held at Hotel Statler, Boston, on Tuesday, April 26, beginning at 10:00 a. m. The speaker at the morning session will be Dr. John A. Kiernan, Chief, Tuberculosis Eradication Division, Bureau of Animal Industry, United States Department of Agriculture. The speaker at the luncheon session will be Dr. James S. Stone, President, Massachusetts State Medical Society.

Reports from the Executive Secretary and Educational Secretary were received.

Resolutions touching upon the death of Dr. Charles E. Marshall, a Vice-President of the Hampshire County Public Health Association, an affiliated organization of the League, were adopted.

It was announced that Miss Ethel L. Dill, R.N., had become a member of the Field Staff of the League and was assigned to work in connection with the Ten Year Program clinics of the State Department of Public Health.

#### BOSTON MEDICAL HISTORY CLUB

A MEETING of the Boston Medical History Club was held March 25th, 1927, in the Boston Medical Library. Twenty members were present:

The first paper of the evening was read by Dr. H. Morrison on "Ludwig Traube." Dr. Morrison spoke of Traube's early studies, on lung changes following section of the vagus nerve (1846), on the pathology of fevers, and on the relation between cardiac and renal disease. Traube had great difficulty in gaining recognition because he was a Jew, but he finally obtained a clinic at the Charité, in Berlin, where he taught with great success and was made professor in 1857. He should be remembered for introducing the clinical thermometer into medicine and, especially, for his founding of the first journal of experimental pathology (1871). His collected works appeared in three volumes in the same year.

Traube was the foremost clinician of his time, and unexcelled in the description of disease. His

fame rests, however, not on his clinical contributions, but on his work in experimental pathology. He insisted on the experimental method of investigating disease.

For many years he suffered from Basedow's disease and died dramatically, surrounded by his faithful pupils.

Dr. Farlow spoke of Traube's picture, owned by the Boston Medical Library, and reproduced in Garrison's History of Medicine.

The second paper, on "Bleeding and Leeching," was presented by Dr. Townsend W. Thorndike. After speaking of the "bleeding shops," common about 1800 and much frequent in the Spring as a more or less social event, by artists and authors, Dr. Thorndike said that bleeding was as old as trephining. Phlebotomy was used in the time of Podalirius, according to Homer, and one finds frequent references to the practice in classical literature. Cupping also was known to Hippocrates, but not leeching. Both Celsus and Antyllus described cups made of bronze, horn or even glass. Scarification is mentioned by Oribasius. In the Middle Ages bleeding was recommended for all diseases and phlebotomy reached its height at the time of the School of Salerno, as described in the "Regimen."

Dr. Thorndike then spoke of the barbers, whose duty it was to bleed, cup, leech, shave, give enemas and do dentistry. Blood-letting was extraordinarily common, also, in the 17th and 18th centuries. Patin bled patients seven to twenty times; Sydenham used it frequently; Rush, in this country, was a strong advocate of the practice, and in Couvresart's wards in Paris, it is reported that 30 out of 112 patients were bled. Dr. Thorndike, also, spoke of leeching, which is still practiced in this country.

Some interesting illustrations of bleeding, leeching and cupping were shown on the screen and various forms of cups were exhibited.

Dr. J. Bapst Blake showed a picture of George Washington in his last illness, attended by two physicians. The picture is now in the Boston Athenaeum.

## THE ANNUAL MEETING OF THE MASSACHUSETTS HOMEOPATHIC MEDICAL SOCIETY

This meeting was held at the Evans Memorial, April 12, 1927. The officers elected for the ensuing year are: Dr. Cecil W. Clark of Newton, President; Vice-Presidents, Dr. William D. Roland and Dr. Charles Colgate; Recording Secretary, Dr. Nathan H. Garrick; Corresponding Secretary, Dr. Conrad Wesselhoft; Treasurer, Dr. Milo C. Green; Chairman of the Board of Censors, Dr. E. S. Calderwood.

Addresses were delivered by Dr. L. B. Boyd head of the Department of Internal Medicine and Homeopathy of the New York Homeopathic

Medical College and Flower Hospital, Dr. J. Emmons Briggs, Dr. Wilson F. Phillips and Dr. Winfred Overholser.

The annual banquet was presided over by Dr. Clifford D. Harvey, the retiring president.

## MEETING OF THE NORTH SHORE MEDICAL FRATERNITY

DR. HAROLD C. BEAN addressed the members of the North Shore Medical Fraternity at their monthly meeting on April 7th, on Back Conditions and Their Treatment.

## ESSEX SOUTH DISTRICT MEDICAL SOCIETY

A REGULAR meeting of the Essex South District Medical Society was held at the Danvers State Hospital, Wednesday, April 6th, 1927. Clinic at 4 P. M. by members of the Staff.

### PROGRAM

Dr. W. F. Wood, Mental Deficiency.

Dr. C. L. Clay. (1) General Paresis with remission following treatment; (2) Demonstration of Tube Feeding.

Dr. Gerald Houser, Dementia Precox (Paranoid Type).

Dr. Heber Teney, Dementia Precox (Problems of care and uncertainty of prognosis).

Dr. Guy C. Randall, Manic Depressive Psychoses.

Supper at 6 P. M. was followed by the speaker of the evening, Dr. Allan W. Rowe, Chief of Research Service of The Evans Memorial, Boston, who spoke upon "The Differential Diagnosis of Endocrine Disorders."

Discussion by Dr. W. F. Wood of Hathorne and Dr. George M. Kline of Beverly.

General discussion from the floor.

W. M. T. HOPKINS, Reporter.

## SOCIETY MEETINGS

### DISTRICT MEDICAL SOCIETIES

#### Essex North District Medical Society

Wednesday, May 4, 1927—Annual meeting. Russell Hall, Young Men's Christian Association Building, 40 Lawrence Street, Lawrence.

Thursday, May 5, 1927—Censors meet for examination of candidates at Hotel Bartlett, 95 Main Street, Haverhill, at 2 P. M.

#### Essex South District Medical Society

Thursday, May 5, 1927—Censors meet for examination of candidates at the Salem Hospital, 3:30 P. M.

Wednesday, May 11, 1927—Annual meeting. The Tavern, Gloucester. Speaker and subject to be announced later.

#### Norfolk District Medical Society

Below are the proposed meetings of the Norfolk District for the remainder of the year. Minor changes may be made in case of necessity.

May 10, 1927—Annual meeting. Details of meeting to be announced.

#### Suffolk District Medical Society

Meetings of the Suffolk District Medical Society and the Boston Medical Library will be held at the Boston Medical Library, 8 The Fenway, Boston, at 8:15 P. M., as follows:

April 27, 1927—Annual meeting. Election of officers. "Medical Education in the Orient and Occident," Dr. David L. Edsall, Dean, Harvard Medical School.

Notices of meetings must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.



## CENSORS' MEETING

THE Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, May 5, 1927, at 4:00 o'clock.

Candidates should make personal application to the Secretary and present their medical diploma at least one week before the examination.

ARTHUR H. CROSBIE, *Secretary*.

520 Commonwealth Avenue, Boston.

## ANNUAL MEETING OF THE NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY

THE National Association for the Study of Epilepsy will hold its next annual meeting at Cincinnati, Ohio, on May the 30th and 31st, 1927, immediately preceding and in joint session with the American Psychiatric Association. Papers will be read by Doctors Bass of Texas, Syz of Baltimore, Clark of New York, Pollock and Davis of Chicago, Shanahan of Sonyea, Notkin of New York, Sharp of Buffalo, Odell of Clifton Springs and others.

G. KIRBY COLLIER.

## BOOK REVIEWS

*The Tired Child*. By MAX SEHAM, M.D., and GRETE SEHAM, Ph.D. J. B. Lippincott Company, Philadelphia and London, 1926. Illustrated, 342 pages.

This volume might have been made into three books any one of which would have been legitimate. It contains a large but not particularly well-selected bibliography. It includes a series of valid medical case histories, and contains in addition a number of pages of "human interest" material like the following:

"An old young face was pressed to the window glass. Sad brown eyes were looking at the black bird with the bright red chest. David wished that he, too, were a black bird with a bright red chest," etc., for four pages when after music lessons, too eagerly persisted in, "A piercing cry had shattered the peace of the night," and finally "The doctor had come and gone. Nervous breakdown. Overstrain. Rest, absolute rest."

To the reviewer this type of volume carries its own death sentence. Unquestionably there are parents who need to be shocked into action, but anyone intelligent enough to read the hundreds of foreign and domestic references will not be edified by the horrid tale of the little black bird with the bright red chest. On the other hand, it is fair to state that most of the emotional incidents are fairly well segregated from

the valid information and the useful suggestions which form the bulk of the book.

*A Manual of Materia Medica for Medical Students*. By E. QUIN THORNTON, M.D., Assistant Professor of Materia Medica in the Jefferson Medical College, Philadelphia. Second Edition, thoroughly revised. Octavo, 384 pages. Lea & Febiger, Philadelphia, 1927.

This book, as is stated in the preface, represents in large part a condensation and adaptation of the material of the tenth (1926) revision of the United States Pharmacopoeia to meet the needs of students and teachers of materia medica. It includes reference to all articles of the Pharmacopoeia, prefaced by a carefully written section of 79 pages of Posology, Prescription Writing, Principles of Medical Combinations and Incompatibilities. In courses where students are required to study the whole content of the Pharmacopoeia it will unquestionably prove of great assistance. On the other hand, in connection with courses where the plan is to study more intensively a smaller list of drugs, this book will be less useful. Not only does the large number of little used drugs introduce confusion. A more serious short-coming is that the discussions of the more important drugs are inadequate. For instance, as to Digitalis there is nothing to indicate the principles now regarded as essential to effectual administration, the last words being simply "Dose.—0.1 Gm. 1½ grains." No doubt the fault lies with the Pharmacopoeia rather than with the author who has abstracted it, but it must be concluded, as a result, that to obtain an understanding command of the best use of drugs the students will have to turn to other sources of information.

*Outlines of Common Skin Diseases*. By T. CASPER GILCHRIST, M.D., Clinical Professor of Dermatology, Johns Hopkins University. Williams & Wilkins Company, Baltimore, 1927. pp. 54.

This is a "tabloid" handbook of dermatology that medical students taking section work in that specialty will find very useful. The physician can make use of it as an amplified index of the presenting locations and symptoms of the common skin diseases. A feature is the small but particularly clear and well chosen photographs that are grouped seven to twelve on a page.

*The Business of Operations*. By JAMES RADLEY. The Digest Publishing Co., Cincinnati, 1927. 96 pages.

The patient's point of view, told in a breezy style. The same subject has been much better covered by previous authors.